TEXAS REPORTS on BIOLOGY and MEDICINE

VOLUME 14, NUMBER 1 SPRING, 1956

Copyright 1956 by the Board of Regents of The University of Texas

Contents

PAGE

An Experimental Study of the Lethal Synergism Between Secobarbital and Alcohol, *John D. Archer* 1

A Method for Serially Sectioning the Knee Joint of the Mature Rat, *James Keith Butler, G. W. N. Eggers, and E. Burke Evans* 6

The Persistence (Survival) of Microorganisms: II. In the Body and Body Materials, *Frank B. Engley, Jr.* 10

Historical Aspects and Present Day Status of the Radical Breast Operation for Cancer, *Joseph K. Johnson* 65

The Effect of Calcium on Chromogenesis in *Micrococcus Pyogenes*, *James A. Johnson* 74

Participation of the Allergen-Specific Enzymatic Factor in Allergic Skin Reaction, *Eugene L. Nowicki* 79

Calling Attention To 90

Founded by Chauncey D. Leake, 1943

EDITOR: John G. Sinclair

ASSISTANT EDITOR: Mary Jane Steding

EDITORIAL BOARD:

Kenneth Earle, Arild Hansen, Grace Jameson, Wiktor Nowinski, Arthur Ruskin

AN EXPERIMENTAL STUDY OF THE LETHAL SYNERGISM BETWEEN SECOBARBITAL AND ALCOHOL*

JOHN D. ARCHER†

The fact that barbiturates and alcohol act synergistically in producing certain effects has been shown experimentally by several investigators (Olszycka, 1935, 1936; Dille and Ahlquist, 1937; Jetter and McLean, 1943; Ramsey and Haag, 1946; Sandberg, 1951; Fearn and Hodges, 1953). Officials of law enforcement agencies have long dealt with the increased severity of intoxication resulting from a combination of barbiturates and alcohol as contrasted with a similar dose of one drug alone. Clinical observers continue to report a synergistic action in the lethal toxicity of the two drugs. The question arises from these reports, however, about whether there occurs a potentiating synergism between the drugs or only an additive synergism. In other words, a question arises about whether the effect of the two drugs acting together is greater than the expected effect of a simple sum of the actions of the two drugs acting in a similar manner.

Jetter and McLean (1937) apparently were the first to call attention to the synergism in toxic effects upon man. They, as well as Burrows (1953), called particular attention to the rapidity of death from combinations of the drugs as compared with death from poisoning by either drug alone. Recent annotations (1953) in the *British Medical Journal* and the *Lancet* discuss deaths from combinations of the drugs in doses that undoubtedly would have been innocuous if taken alone. Interestingly, these annotations were published on the same day, and both reviewed the experimental literature, but the one in the *British Medical Journal* seemed to favor the view that a true potentiation occurs, whereas the one in the *Lancet* favored the view that the synergism was only additive.

* From the Department of Pharmacology and Toxicology, The University of Texas Medical Branch, Galveston. Accepted for publication, September 2, 1955.

† Present address: Student Health Service, The University of Texas, Austin.

Experimentally with rats and mice, Olszycka (1935, 1936) found that alcohol greatly prolonged the sleep induced by butethal. Dille and Ahlquist (1937) worked with rabbits and similarly reported a potentiation between alcohol and pentobarbital upon duration of sleep. Ramsey and Haag (1946) found a prolonged duration of barbiturate-induced sleep in mice when alcohol was given and also found that alcohol decreased the anesthetic dose of thiopental sodium in dogs and prolonged the anesthesia. Sandberg (1951) reported that six barbiturates definitely potentiated the action of alcohol as measured by time of induction of anesthesia in mice. These above reports refuted an earlier report by Carrière, Huriez and Willoquet (1934) that alcohol antagonized phenobarbital-induced coma.

Although this increased duration of depressant action from the combination of barbiturates and alcohol has been frequently observed, the phenomenon does not answer whether a potentiating synergism exists for lethal toxicity, for which there is some clinical evidence. Jetter and McLean (1943) and Ramsey and Haag (1946) demonstrated that sub-lethal doses of alcohol definitely increased the lethal toxicity of barbiturates. Fearn and Hodges (1953) studied this synergism in a more quantitative way and found an additive synergism but not a potentiating one between amobarbital and alcohol as measured by lethal toxicity to mice. Their data, however, indicate the synergism to be approximately a total summation of the toxic action of the two drugs. And as Sandberg (1951) has pointed out, the work of Ramsey and Haag (1946) showed that alcohol had a greater influence on the toxicity of shorter acting barbiturates than upon that of longer acting ones. Since secobarbital is a considerably faster acting drug than amobarbital, it seemed worth-while to investigate quantitatively its toxic synergism with alcohol.

Experimental. Adult Swiss-Webster mice from the colony of the Thomas L. Euers Farm, Austin, Texas were used exclusively in this experiment. The LD_{50} was determined for secobarbital sodium and for ethyl alcohol by the method described by Miller and Tainter (1944). All solutions were administered intraperitoneally. The aqueous solutions of secobarbital sodium were adjusted so that one milliliter of solution was administered per one hundred grams of mouse. The alcohol was administered as a

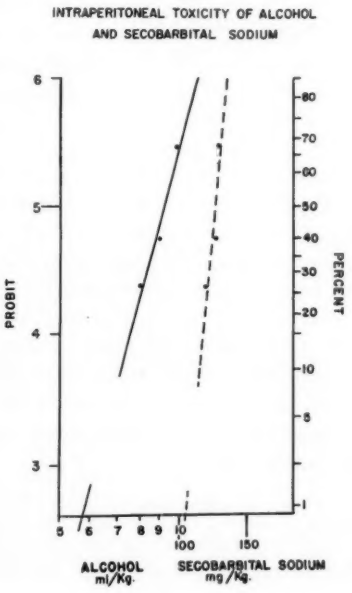
twenty per cent aqueous solution of absolute alcohol. The toxicity data for determining the LD_{50} of each drug are indicated in table 1. These data were plotted on probit paper. As seen in figure

TABLE 1

Number of Animals	Secobarbital Sodium mg./kg.	Alcohol ml./kg.	Number of Animals Dying	Number of Animals Surviving
15	120	..	4	11
15	125	..	6	9
15	130	..	10	5
15	...	8	4	11
15	...	9	6	9
15	...	10	10	5

I, the LD_{50} of secobarbital sodium was found to be 126.5 ± 1.9 mg./kg.; the LD_{50} of alcohol was found to be 9.3 ± 0.4 ml./kg. The ordinate at the right of figure I labelled "Per cent," represents the percentage of mice dying among the total mice treated at the points noted.

FIGURE I



One-half the LD_{50} of each drug was administered intraperitoneally to thirty mice. These doses were 63.25 mg./kg. of secobarbital sodium and 4.65 ml./kg. of alcohol. As seen in figure I, these doses were less than the LD_1 of either drug alone. As shown in table 2, however, nine of the thirty mice died.

TABLE 2

30	63.25	4.65	9	21

Discussion. This study confirms the findings of Jetter and McLean (1943), Ramsey and Haag (1946) and Fearn and Hodges (1953) that barbiturates and alcohol synergize in lethal toxicity. Less than the LD_1 of secobarbital sodium combined with less than the LD_1 of alcohol produced a mortality of thirty per cent in mice. Since on a weight basis, these doses were one-half the LD_{50} of each drug, the synergism involved appears to be an additive type of synergism instead of a potentiating one. Although at the five per cent level of statistical significance, nine deaths in thirty animals do not quite vary significantly from more than fifty per cent deaths (Mainland and Murray, 1952), the results suggest strongly that if any potentiating action should exist, it must be slight. The synergism in lethal toxicity seems to be an additive one and apparently, though not conclusively, results in slightly less than a total summation of the toxic actions of the two drugs.

It should be emphasized that a failure to show a routine and predictable potentiating action between barbiturates and alcohol in laboratory animals by no means rules out the possibility of an occasional such action as observed clinically. As previously discussed, deaths from such a phenomenon apparently do occur in an occasional susceptible individual. The rapidity of at least some such deaths as contrasted with the usual deaths from barbiturates or alcohol alone further emphasizes that in a susceptible person, the toxicity of a combination of the drugs may be greater than could be explained from a simple summation of the expected effects of each drug.

Summary

The existence of a synergistic action between secobarbital sodium and ethyl alcohol in lethal toxicity to mice is confirmed.

Apparently this synergism is additive instead of potentiating and probably involves slightly less than a total summation of the theoretically expected toxic actions of the two drugs. Attention is called, however, to the clinical evidence in the literature that a potentiating synergism may occasionally occur in a susceptible individual.

REFERENCES

Annotations. *British Med. J.*, 1: 1269–1270, 1953.

Annotations. *Lancet*, 264/1: 1140, 1953.

Burrows, M. B.: Alcohol-Barbiturate Synergism. *South African Med. J.*, 27: 1057–1059, 1953.

Carrière, G., C. Huriez, and P. Willoquet: Étude Expérimentale des Injections Intraveineuses d'Alcool au cours d'Intoxications par le Gardénal. *Compt. rend. Soc. de Biol.*, 116: 188–190, 1934.

Dille, J. M., and R. P. Ahlquist: The Synergism of Ethyl Alcohol and Sodium Pentobarbital. *J. Pharmacol. and Exp. Ther.*, 61: 385–392, 1937.

Fearn, H. J., and J. R. Hodges: Synergic Effects of Amylobarbitone Sodium and Ethanol. *J. Pharm. and Pharmacol.*, 5: 1041–1044, 1953.

Jetter, W. W., and R. McLean: Poisoning by the Synergistic Effect of Phenobarbital and Ethyl Alcohol. *Arch. Path.*, 36: 112–122, 1943.

Mainland, D., and I. M. Murray: Tables for Use in Fourfold Contingency Tests, *Science*, 116: 591–594, 1952.

Miller, L. C., and M. L. Tainter: Estimation of the ED_{50} and Its Error by Means of Logarithmic-Probit Graph Paper. *Proc. Soc. Exp. Biol. and Med.*, 57: 261–264, 1944.

Olszycka, L.: Étude Quantitative des Phénomènes de Synergie. Potentialisation de l'Action Hypnotique chez la Souris. *Compt. rend. Acad. Sc.*, 201: 796–797, 1935.

Olszycka, L.: Étude Quantitative des Phénomènes de Synergie. Contribution à l'Étude du Méchanisme des Phénomènes de Potentialisation de l'Action Hypnotique chez le Rat. *Compt. rend. Acad. Sc.*, 202: 1107–1109, 1936.

Ramsey, H., and H. B. Haag: The Synergism between the Barbiturates and Ethyl Alcohol. *J. Pharmacol. and Exp. Ther.*, 88: 313–322, 1946.

Sandberg, F.: A Quantitative Study on the Alcohol-Barbiturate Synergism. *Acta. Physiol. Scand.*, 22: 311–325, 1950.

A METHOD FOR SERIALLY SECTIONING THE KNEE JOINT OF THE MATURE RAT*

JAMES KEITH BUTLER, G. W. N. EGGERS, AND E. BURKE EVANS

A method for preparing serial sections of the knee joint of the mature rat is detailed. The technique consists of: 1. Decalcification of the fixed tissue in a mixture of lactic and nitric acid. 2. Alcoholic dehydration followed by clearation with xylene. 3. Paraffin infiltration in vacuo. 4. Soaking of the trimmed, blocked tissue in a solution of detergent. 5. Sectioning at 12 μ or less. 6. Rapid spreading of the sections followed by the application of pressure to flatten them. This method has, to date, been used on over seventy joints with uniform success.

In order to study the effects of prolonged immobilization upon the knee joint of the mature rat, it was necessary to develop a technique for serially sectioning this difficult material which would yield uniformly satisfactory results. The method described herein was developed after extensive experimentation with celloidon, double imbedding, and numerous variations of the paraffin technique.

This technique is detailed here in an effort to reduce to a minimum the time and frustration likely to be met with by others who may want to prepare this material, and in the hope that the availability of a suitable technique may stimulate more intensive investigation of experimentally produced joint disorders than is reflected in the current literature.

At autopsy, the skin is removed from the leg which, in turn, is disarticulated at the hip joint. The soft tissues are cut to the bone at points about halfway between the knee and hip joints, and halfway between the knee and ankle. The femur, tibia, and fibula are then cut through with a dental separating saw. The use of bone-cutting forceps must be avoided as their application will almost invariably shatter the shaft of the bone.

The tissues are then treated as follows:

1. Helly's fixative ..... 24 hrs.
2. Wash, running tap water ..... 12 hrs.
3. Decalcifying solution .....11–12 days

* From the Orthopedic Research Division, Department of Surgery, The University of Texas Medical Branch, Galveston. Received for publication, August 4, 1955.

The decalcifying solution is constituted as follows:[1]

Water	182 cc.
Lactic acid	14 cc.
Nitric acid, conc.	4 cc.

Each tissue should be placed in at least 150 cc. of the above solution. The decalcifying solution should be replaced on the second and third days after decalcification has begun; for the balance of the decalcification period, the solution should be changed every other day. Each time the solution is replaced, the tissues should be rinsed for two or three minutes in running water. In all cases, the tissue should remain in the decalcifying solution for eleven to twelve days; a shorter period of time will almost invariably lead to unmanageable material.

4.	Wash, running tap water	12 hrs.	
5.	Ethyl alcohol, 70 per cent, 2–3 changes	2	days or more total time

The tissue should remain in the 70 per cent alcohol until it has become firm enough to trim to size. Place the leg upon its side and, with a sharp razor blade, trim it to approximately 1 by 2 cm. Next, place the leg on its freshly cut posterior face and slice away the extreme lateral and medial aspects. This done, the tissue should be about 5 mm. thick and the joint cavity must be open to the surface both laterally and medially.

6.	Ethyl alcohol, 80 per cent,	3 changes	9 hrs. total time
7.	Ethyl alcohol, 95 per cent,	3 changes	6 hrs. total time
8.	Ethyl alcohol, 100 per cent,	2 changes	10 hrs. total time
9.	Xylene	2 changes	5 hrs. total time
10.	Xylene: paraffin,[2]	1:1; 10 inches vacuum	1½ hrs. total time
11.	Paraffin, 25 inches vacuum	2–3 changes	4½ hrs. total time

In order to section properly, it has been found necessary to place the blocked tissue in the microtome and section away the overlying paraffin until the entire side of the tissue lies exposed at the cut face of the block. The block is then placed in a dish containing a 1 per cent solution of Dreft and left on a 45° C. warming table for 24 hours. It may then be replaced in the microtome and sectioned to a depth of 2 to 3 mm. The block is then removed, soaked for another 24 hours, and the remainder sectioned. With reasonable care, only a few sections are lost between the first and second cutting. It should be cautioned that the use

[1] The author is indebted to Mrs. Geraldine Willard, formerly Research Assistant in the Department of Anatomy, The University of Texas Medical Branch, who supplied the formula for the decalcifying solution.

[2] 56–58° C. Tissuemat.

of too concentrated a solution of Dreft or soaking the block too long causes swelling and mushiness of the tissue which will render sectioning impossible.

It is the toughness of the tendinous and ligamentous elements which is minimized by soaking in detergent. Without detergent treatment this material literally comes from the microtome in shreds.

The final problem to be overcome arises from the fact that when heat is applied the various tissues possess rates of expansion differing from that of the paraffin, making it difficult to secure flat sections that are adherent to the slide. For this reason, the commonly employed methods of flattening sections on a water bath or on slides placed on a warming table have not, in our hands, been at all successful. The method described by Gray (1954) has, however, proved admirable.

Slides are coated with Meyer's albumen and a length of ribbon (usually consisting of three sections) is placed upon the slide. With the aid of a pipet, sufficient water to form a sharp meniscus at the edges of the slide is flooded between the ribbon and the slide. The slide is then cautiously moved above the flame of an alcohol lamp until the sections have become completely flat—care being taken to avoid melting the paraffin. The slide is then tipped, the water drained from it, and placed upon the desk top. Next, the sections are oriented with a needle and a strip of wet filter paper extending the length of the slide is placed on top of the sections. The sections are then pressed into complete contact with the slide by rolling a flangeless test tube over the filter paper with considerable pressure. The filter paper is then lifted by one end from the sections. The slides are drained and set aside to dry on a 45° C. warming table. As long as the filter paper is wet when placed upon the sections, there is no danger of the sections' adhering to the paper.

We have used double-edge Durham-type razor blades for sectioning because the edge of a microtome knife rapidly breaks down when cutting this material. Routinely, sections are cut at 12 μ, although, with a sharp knife and modicum of care, we have obtained satisfactory ribbons at 6 μ.

Certain modifications of the above technique may undoubtedly be made with success. The fixing and decalcifying agents were

merely a matter of personal choice and are probably not critical. The use of xylene for clearing, on the other hand, produces results far superior to any of seven other clearing agents tried, and is strongly recommended. Infiltration in vacuo, though probably not essential, is, in this case, superior to and much shorter than ordinary infiltration. The detergent treatment and technique of mounting sections should be closely followed.

REFERENCE

Gray, Peter: *The Microtomest's Formulary and Guide*, Blakiston and Company, Incorporated, New York, 118–119, 1954.

THE PERSISTENCE (SURVIVAL) OF MICROORGANISMS: II. IN THE BODY AND BODY MATERIALS

FRANK B. ENGLEY, JR.*

The importance of the persistence of microorganisms in the body materials during life and after death has been recognized as a major problem and hazard for a long time. The disposal of body excreta, tissues and carcasses themselves have been influenced by the realization that potentially pathogenic organisms might survive in them for long periods depending on certain factors of nature.

The handling of blood, tissues, and other specimens in laboratories and hospitals require precautionary measures since some pathogens may remain alive for a short time and others survive for long periods. The length of survival affects culture procedures for isolation of the organisms since the organisms may die off readily. On the other hand, some organisms are kept in blood or tissue in the laboratory because the organisms survive in them for a very long time.

A distinct hazard and problem is the persistence of pathogens in secretions and excretions such as sputum, urine or feces. The proper disposal methods depend on organisms survivals and persistence in these materials.

Of interest also is the survival of animal, plant and human pathogens in meats and meat products or on plants or plant products which are to be handled or eaten by susceptible individuals or which are to be disposed of without danger to other susceptibles.

The factors involved in the survival and persistence of microorganisms in the body and body materials may be divided into (1) body or body material factors (2) factors in nature and (3) organism factors.

Body or Body Material Factors Affecting. Microorganisms may

* From The University of Texas Medical Branch, Galveston. This study was carried out under contract for Camp Detrick, Frederick, Maryland. Present address University of Missouri, School of Medicine, Columbia, Missouri.

survive in body tissues, fluids or carcasses for varying lengths of time. The body materials may on one hand provide food and protection from outside influences or on the other, may through pH of stomach or of intestine, antagonistic organisms in the intestinal tract or antibodies or other antibacterial factors destroy the pathogens. Some organisms will survive for long periods in whole blood or serum at low temperature. Other organisms will be in tissues and be viable for long periods. Some organisms expelled in urine and feces are not destroyed readily by pH and antagonistic organisms but will be protected and survive. In sputum some organisms are destroyed rapidly but may survive well when suspended or dried outside the body. Body fluids through protective colloids are able to allow organisms to withstand forces of nature.

Forces of Nature or Environment Affecting. Most organisms will persist for longer periods at lower temperatures. At temperatures high enough to allow metabolism to take place, the organisms will grow but kill themselves sooner with detrimental end-products such as acids, aldehydes and other substances. The amount of radiation will affect organism survival, depending on the amount of protection the cells have by organic matter. Wind and rain can wash away and dilute the organisms. Freezing and thawing also may affect the survival of number of cells as well as the heat of the sun if the organisms are near the surface. Another factor affecting survival of organisms in body materials in nature would be the RH if exposed to air. The interplay of temperature and RH is important. If not at soil surface, then the depth at which the body or body material is buried will affect survival.

Organism Factors Affecting. The general and specific characteristics which are a part of a particular cell will affect its ability to survive in the body and body materials. The virulence of the cell and its ability to invade certain tissues are important in determining its presence and survival there. Its resistance to light and other radiation, heat and cold, freezing and thawing, desiccation and temperature, and humidity changes will determine its survival and persistence. Ability to grow at low temperatures or survive at low temperatures, the presence of protective capsules or a spore stage or a particularly resistant cell wall and protoplasm all are important.

Summary

**Bacillus anthracis* (Table B-1). Survival data suggest 60–90 days in blood either moist or dry; in guinea pig blood at 25–30 C. for 1–9 months or at 5–10 C. up to 159 days; the organism may be found in feces and urine of cases. Skin apparently inhibits the organism. In body tissue, survival is given for several days up to 9 months, depending upon condition.

**Spirochetes* (Table B-2)

Borrelia—Survival is listed at refrigeration temperature for 100 days in blood, at —48 C. for 27 months; in feces for 4 weeks and tissues for 1 year.

Leptospira—Survival in blood for 7 days in dark, in feces for 24 hours but in urine for weeks or months. At refrigerator temperature in tissues survival of 26 days is given but at —20 C. 100 days survival is listed.

Treponema—In blood survival is only for a few hours at body or room temperature but in refrigerator for several days and at —20 to —78 C. suvival of months to 3 years is given. In tissues at 5 C. for several days in bodies. Some tissues may stay with viable organisms for 7–10 days or longer. At frozen conditions —10 C. to —78 C. survival of 2 months to 3 years is given.

Spirillum—Survives 1–5 years frozen and in rabbit blood.

**Brucella* (Table B-3). In blood the organisms may live for 3–6 months or up to 5–9 years depending on reports. In feces, data suggest 100 days in dark or 20 days in manured soil. In a patient the organisms were found in feces during 16th month of disease. Skin apparently inhibits the organisms. In urine survival of 6–30 days is listed. In tissues persistence may be for a month to 7–9 months.

**Microorganisms* (Table B-4)

Clostridium—*C. tetani* found in tissues for 4–6 months and feces for 16 days. Gas gangrene organisms found in wound areas for five years. Botulism organisms found in intestines for 4 months.

Corynebacterium—Diphtheria organisms in blood for 13 years in laboratory. In tissues for 9 weeks to 4 months and in throats for 6 months in virulent form.

Erysipelothrix—Alive in tissues for a month, in sunlight for 10–12 days and in buried carcass for months.

Hemophilus—*H. influenzae* survival in blood for a short period but *H. pertussis* lived for weeks.

Lactobacillus—Alive for over 5 years in blood.

Malleomyces—From blood and urine remained alive 16–27 days.

Microbacterium—Found alive in blood for 5 years.

Proteus—In blood survivals of 3–9 years are reported. On skin survival was better than on filter paper.

Pseudomonas—Five year survival in blood is noted and resistance to drying on skin better than on filter paper.

Serratia—Organisms remained alive for 5 years in blood. Survivals on skin were better than on filter paper. Drying appeared to be important in destruction on skin.

•*Diplococcus pneumoniae* (Table B-5). Studies in blood suggest survival of a few months when dried on surfaces and of 5–9 years in tubes. In sputum, persistence of 4 months is reported; when dried on surfaces, a few hours in sunlight to 30–40 days in dark is reported.

•*Escherichia coli* (and coliforms) (Table B-6). Survival in feces in dark or light, fluid or dried varies with report from 3 days in sun to over a year or two. On skin organisms alive for a few minutes to hours. In urine the organisms may live over 100 days. *Aerobacter aerogenes* in feces 9–16 days, inhibited by normal skin and alive in blood 3– years.

•*Metazoa and Protozoa* (Table B-7).

Bartonella—In blood for less than 3 days. Heating and chilling destroy it rapidly.

Entamoeba—Transient viability in feces stored at high temperatures (a few hours) but up to 14–17 days at low temperature. On skin cysts survive only a few minutes.

Necator—In feces plus urine for 2–3 weeks, in feces in laboratory for 13 months and for 3–7 months in soil. In sunlight destruction was in 1–2 hours for larvae.

Ascaris—In sunlight for a few hours but in fecal soil for days to a few weeks.

Trichuris—In fecal material survival of 14 days to 3 months. Increased temperature destroyed the organism readily.

Trichomonas—In pus for 3 hours and vaginal discharge for over 5 days. In laboratory with gastric mucin survival of 2–4 years.

Trypanosomes—Do not develop in blood of bats at low temperature.

Trichinella—Survives a few hours at sub-freezing temperatures.

Plasmodium—In blood at –-50 to —70 C. for 10–15 days.

•*Molds, Yeasts and Fungi* (Table B-8).

M. audouini—In hair survives for 125–420 days.

Tricophyton—Remains viable in toe scrapings for 300 days.

Coccidioides—In sputum in soil for 30 days as vegetative form and 240 days as parasitic form.

•*Mycobacterium tuberculosis* (Table B-9). In blood survival of 14 days while controls in saline live 10 weeks. In fecal material persistence of a few days to several months in nature and 2 years artificially in fecal material. In pus for 3–4 months and skin for 7 years. Sputum samples vary in viability from 1–7 days to over 180 days depending on temperature, light and humidity. In urine it lived for several months. In tissues death resulted rapidly in light and when dried but at low temperatures and if buried survival of 90–167 days reported or even 1–3 years in lung tissue.

•*Micrococcus Species* (Table B-10) (Staphylococcus). In blood for 9–19 years. In feces the effect of sunlight is negligible. On skin drying seems to be the antibacterial factor; low pH affects some while the presence of dirt or fat seems to protect organisms. In pus organisms survive at room temperatures for 2.5–3.5 years with no loss in pathogenicity. The organisms resist pH changes considerably. *Gaffkya* may live in blood for 5 years, *Sarcina* are killed rapidly in throat.

**Neisseria* (Table B-11). The gonococcus stays alive in serum for 7–8 weeks to 16 month. In urethral discharges the organism lives for a few hours at room temperatures. Reduced temperature gives longer survival. In body it may live almost 3 years. The meningococcus may live in nasopharynx for an average of 6 months. In dried secretions viability of several days is reported. In blood the Neisseria live 6 weeks to 3 months.

**Pasteurella* (Table B-12). The plague organism remained viable for 100 days in blood, 3 months in urine, in tissues reports of several weeks in carcasses to 1–2 years in refrigerator to 7 years glycerinated at —15 C. Frozen tissues give 6–42 month survival of *P. tularensis* and up to 10–13 years in glycerinated tisues at —14 C. Pasteurella may live in feces for a few days in nature to several weeks experimentally.

**Rickettsia* (Table B-13). Certain of the rickettsia exist for only 12 days in blood refrigerated, others for 95 days at —70 C. and others for 610 days. In feces survival of 6 years is reported. In tissues at freezing temperatures viability of nearly a year is suggested, for almost 2 years with another while at 5 C. 2–3 months survival is recorded.

**Salmonella Species* (Table B-14). In blood under lab conditions survival of 7 years is reported. In feces, 8 days to over 8 months is listed. On skin survival of 10–20 minutes on clean skin to several hours on dirty or fatty skin is suggested. Frozen turkey skin harbors the organism for over a year. In tissues the organisms withstand heat for short periods.

**Salmonella typhosa* (Table B-14). In blood for at least 7 years, one strain for 18 years, others up to 10 years in virulent state. In feces for a few days to 5 months with varying conditions. The normal skin does not allow survival but for a short time. In urine for 2–3 days at high room temperatures to 14 weeks. In tissues existence for 140–160 days is suggested.

**Shigella* (Table B-15). In feces under varying conditions survival of a few hours on fruit to over 200 hours in desert to several days and even up to 113 days in dried feces. In urine at room temperatures up to 40–50 days. Gastric juice was germicidal.

**Streptococcus* (Table B-16). In blood survival of a few weeks (4–8) to 7–19 years is recorded. On skin streptococci survive for 1–2 hours. In sputum viability may be as long as 150 days. In tissues 3 month to 6 month survival is recorded.

**Vibrio* (Table B-17). In blood the organism exists for 47 hours to 8 days and longer (5 weeks) in blood broth. In feces under adverse conditions of pH and sun and temperature viability varies from 24 hours to 30 days. In urine extremes of 6–40 days are recorded.

**Viruses* (Table B-18).

Hoof and Mouth—In blood and serum at low temperatures for several weeks to months for survival and in lymph existence for over 2 years is listed. In feces survival of 2 months to almost a year is suggested at low temperature. In tissues viability of the virus remained for 2–5 months at refrigerator temperatures.

Herpes—Alive only 40 minutes in normal rabbit serum, 10 minutes in serum plus UV. In brain suspension it lived for 100 hours.

Yellow fever—In blood it was viable for 154 days when frozen but in blood and liver at —10 C., 2 weeks.
Rift Valley—Long viability in refrigerator at 82 days or 2 years. In serum survival at refrigerator temperature was longer—1048 days.
Infectious jaundice—In dried fecal dust, the virus lived for 31 days.
Newcastle—The virus was present in chicken feces. On skin and carcass 96 days and in bare and unplucked carcass, 134–196 days viable.
Psittacosis—In fecal material the virus remained for 10 days.
Poliomyelitis—Fecal material harbored the virus for hours after passage. Storage at low temperature protected the virus to allow survival of 7–8 weeks up to 6 months. Virus found in stools from 7th day of disease to 123 days following attack. In tissue survivals of 20–30 days reported, in nasopharynx for a number of days (5–9) after onset of disease. The virus remained viable in an amoeba culture for less than 3 days.
Rabies—In brain material viability of 47 to over 68 days is reported. Exposed to liquid air destroyed in 24 hours and at high and low pH levels in a few hours.
Influenza—On human skin the virus was destroyed in less than 1 hour. In tissues at —30 C. survival was less than 6 months, lower temperature of —78 C. protected for 6 months in broth and in rabbit testes for 3 years.
Fowlpox—Two year survival in dried lesions reported.
Vaccinia—The virus from pustules survived up to 8 hours. In mouse brain survival of 6 months to 2 years is noted. In calf lymph equal survival is given.
Smallpox—In dried crusts, the virus remained for periods over a year in the light and dark.
Rinderpest—In rabbits and storage viability of 7 days.
Encephalitis viruses—Storage of 1 year in 50 per cent glycerin, loss of virulence on drying. If frozen, survives over 3 months. Jap B in mouse brain survives at —78 C. for 6 months.
Lymphogranuloma inguinale—In rabbit testes survival of 10 months at —78 C. is reported.
Pneumoenteritis—Loss of virulence on storage is listed at 6 days if frozen and in 20 days if dried and refrigerated.

TABLE B1

The Survival of Bacillus Anthracis in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
Blood, R.T., moist spores	60–90 d.	Minett	1950
Blood, R.T., dry	60–90 d.	Minett	1950
G.P. blood, 25–30 C., opened before decomposition	1–9 mos.	Stein	1947
G.P. blood stored 5–10 C.	35–159 d.	Stein	1947
FECES			
G.P. feces	Spores in 7 of 19	Stein	1947
SKIN			
100% inhibited by normal human skin		Hill	1933

URINE			
Urine of G.P.	Vegetative forms in 19 of 28 cases	Stein	1947
TISSUES, GENERAL			
Muscle, G.P., 25–30 C., opened before decomposition	6–7 mos.	Stein	1947
Liver and bone marrow, G.P., 25–30 C., opened before decomposition	6 mos.	Stein	1947
Spleen, opened before decomposition, R.T.	3 mos.	Stein	1947
Stored 5–10 C.	14–24 d.	Stein	1947
Continuous freezing, spleen, —60 to —70 C., G.P.	Destroyed in 90 d.	Stein	1947
Carcass of unopened G.P., 25–30 C.	Few alive in 72–80 h.	Stein	1947
Carcass of unopened G.P., 5–10 C.	4 wks.	Stein	1947
Carcass of unopened G.P., R.T.	9 mos.	Stein	1947

TABLE B2

The Survival of Borrelia, Leptospira, Spirillum and Treponema Species in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
B. recurrentis			
Defibrinated, refrigerated sheeps blood	195 d.	Beck	1937
Infected blood, Inoc. into physiol. saline with boiled egg white	Still viable 3–4 wks.	Krantz	1925
At ice box temp.	Still viable 4 d.	Krantz	1925
Rat blood, —48 C.	Remained infective for mice 27 mos.	Lofgren	1945
Iced blood	1 wk.	Toyoda	1931
Rat blood, —12 to —20 C.	Infectivity markedly reduced, 6 wks.	Turner	1939
Rat blood, —78 C., Cooling	2–6 h.	Turner	1939
—78 C. to 0 C.	2–6 h.	Turner	1939
Clotted blood, R.T.	6 d.	Wynns	1935
0 C.	100 d.	Wynns	1935
L. icterohemorrhagica			
Blood, defibrinated, R.T., light of day, 45 C.	Still infective 7 d. Still infective 15 min.	Uhlenhuth	1916 1916
S. minus			
Frozen	1 yr.	Turner	1939
S. rubrum			
Sterile rabbit blood susp.	at least 5 yrs.	Frobisher	1947
T. pallidum			
Citrated blood and plasma stored at 5 C.	Occasionally at 48 and 72 hrs. Not after 72 hrs.	Bock	1941
Human blood and plasma, 5 C.	72 h.	Kolmer	1942
Serum exudate from chancre, R.T.	121 d.	Lacy	1921

BLOOD, (Continued)			
10% rabbit serum, —20 C.	2 mos.	McLeod	1949
Frozen plasma, —78 C.	3 yrs.	Ravitch	1942
Blood, ice box	96 h. non-infectious	Ravitch	1949
Plasma, —20 C.	>48 h., non-infectious	Ravitch	1949
Rabbit plasma, 5 C.	6 d.	Selbie	1943
Blood of G.P., 14 C.	2–33 d.	Sergent	1938
Blood of G.P., 20 C.	33–60 d.	Sergent	1938
Blood of G.P., 0 C.	7–8 wks.	Sergent	1938
Blood of G.P., 10 C.	1 d.	Sergent	1938
Citrated whole blood, 5 C.	72 h.	Turner	1941
Plasma, 5 C.	62 h.	Turner	1941
Human serum, physiol. saline under vaseline, R.T.	5 d.	Zurhelle	1927
In organ parts, R.T.	3 d., 17 hrs.	Zurhelle	1927
FECES			
Borrelia spp.			
Feces	4 wks.	Gowen	1945
L. icterohemorrhagica			
Feces	24 h.	Noguchi	1918
Urine, body temp.	wks. or mos.	Sawers	1938
URINE			
L. icterohemorrhagica			
R.T.	Still infectious 2 d.	Uhlenhuth	1916
TISSUES, GENERAL			
B. duttoni			
Mouse tissue, —78 C.	1 mo.	Oag	1939
Frozen rabbit testes, —78 C.	1 yr.	Turner	1939
L. icterohemorrhagica			
Infected G.P. liver in ice	26 d.	Buchanan	1927
Whole pig liver blocks, —20 C., frozen	Viable and virulent 100 d.	Stavitsky	1945
Whole pig liver blocks, lyophilized	0	Stavitsky	1945
Frozen rabbit testes, —78 C.	10 mos.	Turner	1939
Decomposing liver, R.T.	27 h.	Uhlenhuth	1916
Infected liver, R.T.	3 d.	Uhlenhuth	1916
Infected G.P. liver, ice	26 d.	Uhlenhuth	1916
Treponema pallidum			
Dead autopsy tissue, 5 C.	3–5 days after death	Armuzzi	1926
Rabbit testis, heavy susp.	72 h.	Block	1941
Citrated rabbit blood, 3–5 C., mixed with testis	96 h.	Block	1941
Testicular in vitro x-tract.			
39 C.	5 h.	Boak	1932
40 C.	3 h.	Boak	1932
41 C.	2 h.	Boak	1932
41.5 C.	1 h.	Boak	1932
Dead bodies of cong. syphilitic children, refrig.	24–36 h.	Hoffman	1926
Rabbit testes dried in vacuo from frozen state	nil.	Hampp	1951

TISSUES, GENERAL, (Continued)			
Corpse of cong. syphilitic child	5 d.	Koch	1911
Corpse of cong. syphilitic child, dried and incub. 37 C.	Recov. 0 72 h.	Kratzeisen	1923
Liver and sterilized gall.	Recov. 0 72 h.	Kratzeisen	1923
Liver with bouillon	Recov. 0 72 h.	Kratzeisen	1923
Liver with NaCl	Recov. 0 72 h.	Kratzeisen	1923
Liver with water	Recov. 0 72 h.	Kratzeisen	1923
Human autopsy material ice box	48 h.	Lacy	1921
Chancre	7 d.	Lacy	1921
Saline susp., rabbit testicle, R.T.	58 d.	Lacy	1921
Rabbit testicle, ice box	58 d.	Lacy	1921
Rabbit testes, 5 C.	Recov. in 3 d.	Levaditi	1946
Autopsy mat'l. from syphilitic patients, 5 C.	Recov. in 3 d.	Levaditi	1946
Susp. rabbit testes, —78 C.	1 yr.	McLeod	1949
Tissue susp., 37 C.	24 h.	Miyao	1930
Syphilitic mat'l. 10C.	3 h.	Neisser	1911
Syphilitic mat'l. ice chest	24 h.	Neisser	1911
Syphilitic mat'l. 48 C.	30 min.	Neisser	1911
Tissue susp., 0 C.	2 h.	Miyao	1930
Autopsy mat'l. in ice box	7 d.	Rosahn	1935
Rabbit testes	10 d.	Perry	1948
Treponema pallidum			
Frozen exudates	13 mos.	Shpuntoff	1949
Testicular extracts, —10 C.	<2 mos.	Turner	1938
Testicular extracts, —20 C.	<2 mos.	Turner	1938
Testicular extracts, —78 C.	1 yr.	Turner	1938
Testes of rabbit, —78 C.	3 yrs.	Turner	1939
Excised chancre, R.T. dried in vacuum	68 h. to 4 d.	Zurhelle	1927
T. pertenue			
37 C., tissue susp.	2 h.	Miyao	1930
Testicular extracts, —78 C.	1 yr.	Turner	1938
Testes of rabbits, —78 C.	3 yrs.	Turner	1939

TABLE B3

The Survival of Brucella Species in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
B. abortus			
Sterile rabbit blood susp.	At least 5 yrs.	Frobisher	1947
B. bronchiseptica			
Sterile rabbit blood susp.	9 yrs.	Frobisher	1947
Broth culture, 0 C.	43 d.	Hampil	1932
B. melitensis			
Sterile rabbit blood susp.	3 mos.	Frobisher	1947
Normal skin of G.P. more vulnerable to B. melitensis entry than digestive tract		Hardy	1929

BLOOD, (Continued)			
B. spp.			
Citrated 4 C. from convalescing patient	6 mos.	Spink	1950
B. suis			
Sterile rabbit blood susp.	3 mos.	Frobisher	1947
FECES			
B. abortus			
Isolated from patient's stool in 10% CO_2, aerobically and anaerobically	Surviving in 16th month of infection	Amoss	1929
B. melitensis			
Dry, sterile manured soil,	72 d.	Horrocks	1906
Moist, sterile manured soil,	7 d.	Horrocks	1906
Moist, unsterile manured soil,	20 d.	Horrocks	1906
B. suis			
Feces, sterile, dark	100 d.	Anonymous	1933
Feces, sterile, dark	100 d.	Cameron	1933
SKIN			
B. melitensis			
34% inhibited on normal human skin		Hill	1933
URINE			
B. melitensis			
Favorable conditions	6 d.	Bang	1894
Urine contam. with dust	30 d.	Chief	1944
TISSUES, GENERAL			
B. abortus			
Uterus of cow, F.P.	9 mos.	Bang	1897
Uterine exudate, F.P.	7 mos.	Bang	1897
Hog, lymph nodes	Found in packing house	McCullough	1949
Dogs fed aborted fetus, placenta and lymph nodes	Virulent 151 d.	Morse	1951
B. melitensis			
Uterus, F.P.	6 d.	Bang	1897
Colon at autopsy	More prevalent in cold wet season than dry and hot.	Eyre	1908
B. suis			
37–39 C., in vivo	Did not persist for prolonged periods	Braun	1951
Hog tissue, —10 F.	Still viable, 30 d.	Huddleson	1933
Spleen	Recov. small number, 40 d.	Huddleson	1933
Swine tissue, —10 F.	Recov. good percent, 40 d.	Huddleson	1933
Spleen, 40 F.	45 d.	Huddleson	1933

TABLE B4

The Survival of Clostridium, Corynebacterium, Hemophilus, Lactobacillus, Malleomyces, Microbacterium, Proteus, Pseudomonas, Serratia and Erysipelothrix in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
Clostridium tetani			
Free spores from animals dying of tetanus. Inject subcutaneous	Recov. up to 17th d.	Canfora	1907

BLOOD, (Continued)			
May remain dormant at site of injection for as long as 4 mos.		Francis	1914
Corynebacterium pseudotuberculosis (Preisz-Nocard)			
Horse serum in flask, light	11 mos.	Urbain	1930
Horse serum in flask, dark	13 mos.	Urbain	1930
Horse serum in test tube, light	1 mo.	Urbain	1930
Horse serum in test tube, dark	11½ mos.	Urbain	1930
Corynebacterium diphtheriae			
Washed, dried in sunlight (serum)	121 d.	Abel	1893
Sterile rabbit blood susp.	Hundreds of strains survived 13 yrs.	Frobisher	1947
Hemophilus influenzae			
Sterile rabbit blood susp.	6 wks. or less	Frobisher	1947
Blood broth, —15 C.	2½ hrs.	Hampil	1932
Blood broth, —20 C.	1½ hrs.	Hampil	1932
Hemophilus pertussis			
Sterile rabbit blood susp.	6 wks. or less	Frobisher	1947
Lactobacillus acidophilus			
Sterile rabbit blood susp.	At least 5 yrs.	Frobisher	1947
Microbacterium, spp.			
Sterile rabbit blood susp.	5 yrs.	Frobisher	1947
Proteus spp.			
Sterile rabbit blood susp.	3–9 yrs.	Frobisher	1947
Pseudomonas spp.			
Sterile rabbit blood susp.	5 yrs.	Frobisher	1947
FECES			
Cl. botulinum			
Feces	No survival	Burke	1919
Feces	No survival in human and animal following ingestion	Easton	1924
Cl. tetani			
Dog feces	16 d.	Sormani	1891
Malleomyces pseudomallei			
26–27 C., culture	27 d.	Fletcher	1928
1% phenol, 37 C.	24 h.	Fletcher	1928
1% formalin, 40 C.	24 h.	Fletcher	1928
SKIN			
Proteus spp.			
Proteus species survived longer on skin than on filter paper		Hellat	1948
1.6% inhibited on normal skin		Hill	1933
Pseudomonas aeruginosa			
Survived longer on skin than on filter paper		Hellat	1948
4.4% inhibited on normal skin		Hill	1933

SKIN, Continued			
Serratia marcescens			
Skin or arm, cult. dil. 1:1,000, dry	Inoc. 718, Recov. 94 after 5 min.	Arnold	1934
Skin, cult. dil. 1:1,000 moist	Inoc. 696, Recov. 151 after 5 min.	Arnold	1934
Skin of back, not washed	Inoc. 1,000, Recov. 1 after 30 min.	Arnold	1930
Skin, body temp., dirty or fatty	Recov. 0, 30 min.	Arnold	1930
Skin, 37 C., before drying	Inoc. 34,000, Recov. 0 in 15 min.	Bryan	1933
Skin, 37 C., after drying	Inoc. 51,000, Recov. 0 in 10 min.	Bryan	1933
Sterile rabbit blood susp.	At least 5 yrs.	Frobisher	1947
Survived longer on skin than on filter paper		Hellat	1948
16.1% inhibited on normal skin		Hill	1933
Palmar surface, clean hand	Inoc. 1,200, Recov. 0 after 2 min.	Norton	1932
Washed hide	Inoc. 520, Recov. 0 after 20 mn.	Norton	1932
Palms of hands	Inoc. 1,200, Recov. 112 after 1 min.	Norton	1932
Kept moist by water vapor	Inoc. 920, Recov. 1,000 after 30 min.	Norton	1931
Dry surface of skin	Inoc. 770, Recov. 900 after 70 min.	Norton	1931
Skin of forearm	0–40 min.	Norton	1931
Skin, moist with water vapor	Inoc. 310, Recov. 150 30 min.	Norton	1931
Unwashed tanned hide, dry	Inoc. 3,000, Survive 4 hrs. 15 min.	Norton	1931
Washed tanned hide, dry	Inoc. 2,440, Survive 1 h. 20 min.	Norton	1931
Dirty or fatty skin	Several hrs.	Singer	1929
URINE			
Malleomyces pseudomallei			
26–27 C., culture	16 d.	Fletcher	1928
TISSUES, GENERAL			
Clostridium botulinum			
Intestinal tract, 22 C.	4 mos.	Burke	1919
Cl. sporogenes			
War wounds	5 yrs.	Anonymous	1923
Cl. tertius			
War wounds	5 yrs.	Anonymous	1923
Cl. tetani			
Liver, spleen, bone marrow, lungs, lymph glands. Free spores from dying animals	Recov. up to 55 d.	Canfora	1907
Kidney, spleen	Found at site of inoculation after 26 hrs.	Koser	1917

TISSUES, GENERAL, (Continued)			
Cl. tetani			
Horse intestine	4 mos.	Noble	1915
Fresh G.P. feces	16 d.	Noble	1915
Remain viable at site of inoculation	6 mos.	Semple	1911
Recovered from liver, spleen kidney, lungs and bone marrow following subcut. inject.	11 d. to 3½ mos.	Tarozzi	1906
Corynebacterium diphtheriae			
Small dried pieces	9 wks.	Abel	1893
Inoc. on membrane	4 mos.	Abel	1893
Throat of boy after attack	Virulent 6 mos.	Macgreggor	1898
Erysipelothrix rhusiopathiae			
Dried, dark	1 mo.	Hull	1947
Sunlight	10–12 d.	Hull	1947
Salted or pickled meat	3–4 mos.	Hull	1947
Carcass of buried animals	mos.	Losner	1896
Erysipelothrix spp.			
18–20 C., carcass	mos.	Hettche	1937
Serratia marcescens			
Mouth, 28 C.	16 h.	Teague	1912

TABLE B5

The Survival of Diplococcus Pneumoniae in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
R.T., sealed tubes, dark	.....	Arkharow	1892
Broth, saliva, or 0.5% saline susp., R.H. 40–55%, Type I	Very high mortality rate	Dunklin	1948
Broth, saliva, or 0.5% saline susp., R.H. above or below 50%, Type I	Long periods	Dunklin	1948
Inject into G.P., 37 C.	65½ hrs.	Emmerich	1894
Sterile rabbit blood susp.	5 of 8 strains—5 yrs. 11 strains, 9 yrs.	Frobischer	1947
Fluid, defibrinated blood	2 mos.	Gilbert	1896
Rabbit blood dried, 80 F., glass and gauze, light and dark, Type II (smooth)	2 mos.	Stillman	1940
40 F., in ice box, dark, glass and gauze, Type II (smooth)	12 mos.	Stillman	1940
80 F., on glass, daylight, dried, Type I	1 mo.	Stillman	1940
80 F., on glass, dark, dried, Type I	2 mos.	Stillman	1940
40 F., in ice box, dark, Type I			
glass	12 mos.	Stillman	1940
gauze	9 mos.	Stillman	1940
80 F., daylight, glass and gauze, Type III (smooth)	5 mos.	Stillman	1940

BLOOD, (Continued)			
80 F., dark, glass, Type III	7 mos.	Stillman	1940
80 F., dark, gauze, Type III	11 mos.	Stillman	1940
40 F., dark, ice box, glass	13 mos. (Type III)	Stillman	1940
40 F., dark, ice box, gauze	12 mos. (Type III)	Stillman	1940
SPUTUM			
37 C., with dust, air dried	Recov. 0 in 25 d.	Germano	1897
37 C., dried	4 mos.	Guarnieri	1888
Dried in test tubes, 80 F., diffuse daylight, Type III	20 wks.	Stillman	1940
Dried, ice box, 40 F., dark	40 wks.	Stillman	1940
Dried, Type I	4 wks.	Stillman	1940
Dried sputum, dark;	35 d.	Wood	1905
diffuse light	30 d.	Wood	1905
Moist sputum, 22 C., dark;	11 d.	Wood	1905
0 C., dark	35 d.	Wood	1905
Moist sputum, 22 C., strong light	<5 d.	Wood	1905
Dried, sunlight	<4 h.	Wood	1905
Powdered, dark	1–4 h.	Wood	1905
Powdered, sunlight	1 h.	Wood	1905
Sprayed	24 h.	Wood	1905
Moist sputum	<2 wks.	Wood	1905
Dried, dark	35 d.	Wood	1905
diffuse light	30 d.	Wood	1905
GENERAL			
32–35 C., abdominal cavity	4–5 d.	Foa	1888

TABLE B6

The Survival of Escherichia Coli, Aerobacter and Paracolobactrum in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
Normal rabbit serum, drying	Death rate logarithmic so that it is proportional to conc. of surviving bacteria	Chick	1912
M.O. added to normal rabbit serum	Living bact. remain stationary	Chick	1912
FECES			
Cow feces	2–3 h.	Cohn	1950
Fl. stool, R.T., dark	>55 d.	Dold	1944
Stool, dried on filter paper, R.T., dark	>45 d.	Dold	1944
Watery stool, R.T., dark	>143 d.	Dold	1944
Thin watery stool dried on filter paper	>143 d.	Dold	1944
Fluid stool, R.T., dark	6 mos.	Dold	1944
Fl. stool, dried on filter paper, R.T., dark	Recov. pure cult. >8 mos.	Dold	1944
Fl. stool, R.T., dark	>5 mos., 16 d.	Dold	1944
Stool, dried on filter paper, dark, R.T.	2 mos., 21 d.	Dold	1944

FECES, (Continued)			
Fl. stool in dark, R.T.	>3 mos., 19 d.	Dold	1944
Dried stool in dark, R.T.	71 d.	Dold	1944
Fl. stool in dark, R.T.	1 mo.	Dold	1944
Dried stool in dark, R.T.	1 mo.	Dold	1944
Fl. stool, dark, R.T.	>54 d.	Dold	1944
Dried stool, dark, R.T.	>54 d.	Dold	1944
Fecal susp., 20 C.	8 wks.	Jordan	1926
37 C.	20 d.	Jordan	1926
10 C.	22 wks.	Jordan	1926
—11 C.	18 wks.	Jordan	1926
R.T.	2 mos.	Jordan	1926
Feces, —8 F. to —30 F., exposed to open air	>100 d.	Lu-Ti-Huan	1930
Fecal emulsion, dilute, diffuse sunlight	3 d.	McNaught	1910
inside window	11 d.	McNaught	1910
test tube, direct sunlight	19 h.	McNaught	1910
Feces, saline susp., 37 C.	<3 mos.	Parr	1937
Feces, saline susp. ice box temp.	599 d.	Parr	1937
Feces, 37 C.	1 yr., 8 mos.	Parr	1937
Feces, 40 C.	2 yrs.	Parr	1937
Feces, —11 C.	2–3 mos.	Parr	1937
Feces, ice box temp.	359 d.	Parr	1937
Feces, animal, 61 F., inoc. 1–3 thos. 52 F.	Recov. 3–10 in 4 wks.	Savage	1917
Feces, on stump, blizzard inoc. 532,000/gm.;	Recov. 0, 18 d.	Tonney	1931
on stump, spring, inoc. 11,800/gm.;	Recov. 0, 153 d.	Tonney	1931
on stump, warm season; pure cult, stump, winter, inoc. 323,000/gm.	172 d. Recov. 0, 22 d.	Tonney Tonney	1931 1931
SKIN			
Palmar surface, clean, inoc. 4,000	Recov. 1 after 10 min.	Arnold	1930
Skin, body temp. dirty or fatty	Several hours	Arnold	1930
Skin, palmar surface, clean hand, body temp.	Recov. 0 after 10 min.	Arnold	1930
Skin, 37 C., inoc. 78,000	Recov. 0 after 10 min.	Bryan	1933
Wiping hand with towel		Grubb	1947
Survived longer on skin than on filter paper		Hellot	1948
33% inhibited on normal human skin		Hill	1933
Palm of hand	Entirely dest. in 10 min.	Krueger	1942
Skin, desiccation, high R.H.	8 h.	Rebell	1950
99%, 96 F.	2 wks.	Rebell	1950
URINE			
Exposed to open air, —8 F. to —30 F.	>100 d.	Lu-Ti-Huan	1930
GENERAL			
Stomach-absence of free HCl.	Coliforms found in abundance	Lohr	1927
Isolated from frozen shrimp more frequently than any other coliform organism		Holmes	1949

FECES			
Aerobacter aerogenes			
Crude, dil. 1/100,000	Not given	Atkinson	1934
Sterile rabbit blood susp.	3–9 yrs.	Frobisher	1947
Found by many workers in human feces		Gray	1932
Fresh, R.T.	16 d.	Jordan	1926
Pure cult. on stump during blizzard in winter	9 d.	Tonney	1931
SKIN			
Aerobacter aerogenes			
5% inhibited on normal human skin		Hill	1933
BLOOD			
Paracolobactrum aerogenes			
Sterile rabbit blood susp.	3–9 yrs.	Frobisher	1947

TABLE B7

The Survival of Metazoa and Protozoa in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
Anaplasmosis (Bartonella bovis)			
Dried blood	<3 d.	Dypstra	1948
Blood submitted to heating and chilling over period of 6 d.	0	Dypstra	1948
Plasmodium vivax			
—50 to —70 C.	10–15 d.	Saunders	1947
Tryponosoma equiperdum			
Bats kept at low temp.			
10 C. to 3–4 C.	Do not develop	Kalabuchov	1935
33 C. to 37 C.	Develop in blood in short time	Kalabuchov	1935
FECES			
Entamoeba histolytica			
Feces, 37 C.	2–4 h.	Gurevitch	1947
Feces, 25–30 C.	5–8 h.	Gurevitch	1947
Feces, 6–8 C.	8–10 h.	Gurevitch	1947
Infected feces, 27–30 C.	9 d.	Kuenen	1913
Infected feces, 37 C.	3 d.	Kuenen	1913
Infected feces, with 1 cc. salt mixture, freezing inoc. 50 cysts	Recov. 13 dead in some h.	Kuenen	1913
Stool emulsion, R.T., allowed to dry on fingers	10 min.	Spector	1934
Dried feces	Instant death	Wenyan	1917
Very dilute feces	>1 mo.	Wenyan	1917
Feces with free Cl_2 in water, 1:10,000	No effect in several h.	Wenyan	1917
0 C., 16 h. cult.	9 d.	York	1926
16–20 C.	7 d.	York	1926
0 C. washed and conc. cysts	17 d.	York	1926
16–20 C., washed and conc. susp.	10 d.	York	1926

FECES, (Continued)			
Human feces, cult. tube	14 d.	Dobell	1936
R.T. 4 h., and 14 C.for 14 h.	2 d.	Beaver	1949
Soil plus stool plus water R.T., 1 h.	6 d.	Beaver	1949
14 C. 12 h.	4 d.	Beaver	1949
28–34 C.	8 and 4 d.	Beaver	1949
Feces	3 d.	Rudolfs	1951
Entamoeba coli			
Human feces, 1–2 C., cult. cysts	4½ mos.	Dobell	1936
Stool emulsion, R.T., allowed to dry on fingers	More resistant than *E. histolytica*	Spector	1934
Entamoeba coli marcorcarum			
Monkey feces, naturally infected	4½ yrs.	Dobell	1936
Hookworm			
Night soil, dil. with urine, summer temp.	99% killed, 2–3 wks.	Cort	1925
Night soil, dil. with urine, summer temp.	99% killed, 2–3 wks.	Cort	1926
Human feces, direct sunlight	Lethal even in moist tropics	Faust	1924
Moist feces, lab. temp.	13 mos.	Galli	1905
Water, moist feces, larvae	7 mos.	Leichtenstern	1887
Fecal mat'l in abandoned latrine in brickyard	3 mos.	Leichtenstern	1887
60 F., strong sunlight	>2 h. (larvae)	Nicoll	1917
Strong sunlight under glass	1 h.	Nicoll	1917
Necator americanus			
45 C.	2 h.	Nicholls	1939
Ascaris lumbricoides			
Human feces on sand in sun, ova	21 d.	Brown	1927
Human feces on sand in shade	Recov. 90.8%, 35 d.	Brown	1927
Clay in sun	71% motile, 21 d.	Brown	1927
Clay in shade	85% motile, 21 d.	Brown	1927
Loam in shade	89.3% motile, 21 d.	Brown	1927
Human feces in shade	72–92% motile, 4 wks.	Caldwell	1928
Pig feces in shade	99% motile, 4 wks.	Caldwell	1928
Human feces, drying in sunlight, 130 F.	35–40% disintegrated in >7½ h.	Caldwell	1928
Pig feces, sunlight, 130 F.	5–30% disintegrated in >7½ h.	Caldwell	1928
Human and pig feces, lying in shade	Recov. 0, 2 wks.	Caldwell	1928
Human feces moist with constant temp. 40–50 C.	38% ova disintegrated in 14 d.	Caldwell	1928
Pig feces, moist with constant temp. 40–50 C.	40% ova disintegrated in 14 d.	Caldwell	1928
45 C. compost	3 mos.	Nicholls	1939
54–55 C.	3–5 min.	Nolf	1932
Fecal susp. of eggs	All degenerate in 27–35 d.	Rudolfs	1951
Trichuris trichuris			
Human feces, 30 C. on cover slip, incubator, moisture, sat'd, inoc. 1,000	Recov. 0, 29 d.	Nolf	1932
—12 C., 4 d. incubation before exposure	6 d.	Nolf	1932
—9 C., 4 d. incubation	Recov. 40%, 8 d.	Nolf	1932

FECES, (Continued)			
Sand-in shade, human	74% motile, 35 d.	Brown	1927
Eggs of *T. trichiura*, as well as *N. americanus*, *A. lumbricoides*, *T. saginata*, *S. haematobium*, pass thru cockroach unharmed		Macfie	1922
URINE			
A. lumbricoides			
Human urine	No survival	Yoshida	1920
Trichomonas vaginalis			
Urine, 20 C., inoc. with vaginal secretion	Few alive in 20 h.	Jirovec	1948
GENERAL			
T. vaginalis			
Vaginal discharge	Recov. 2–4% in 6 h.	Kessel	1950
Vaginal discharge, R.T., paraffin sealed coverslips	>5 d.	Kirby	1943
Pus, dry	3 h.	Swift	1937
E. histolytica			
Cysts drying on human hand	5 min.	Spector	1934
Cysts pass through cockroach unharmed		Macfie	1922
T. batrachorum			
In Riana pipiens, R.T., distilled water, and gastric mucin	6 yrs. and 11 mos.	Wenrich	1949
T. wenyani			
In white rats, R.T., with distilled water and gastric mucin	4 yrs.	Wenrich	1949
In amphiuma, R.T., with gastric mucin	4 yrs. (T. prowazeki)	Wenrich	1949
T. augusta			
In Rana pipiens, R.T., with water and gastric mucin	2 yrs.	Wenrich	1949
T. hominis			
In man, 32–34 C., in water and grstric mucin	3 yrs.	Wenrich	1949
T. species			
From Crotelus horridus	2 yrs.	Wenrich	1949
Trivitus Parva			
From *Triturus viridescens*	4 yrs.	Wenrich	1949
Monocercomonoides			
From Lipulid larvae	4 yrs.	Wenrich	1949
T. spp.			
From Japanese beetle larvae	4 yrs.	Wenrich	1949
Trichinella spiralis			
G.P., rat muscle, pork, pork sausage, —35 C.	2 h.	Blair	1934
—17.8 C.	53 h.	Blair	1934
Tr. spp. (larvae)			
Pork, —27 C.	36 h.	Gould	1949
—30 C.	24 h.	Gould	1949
—33 C.	10 h.	Gould	1949
—35 C.	40 min.	Gould	1949
—37 C.	2 min.	Gould	1949

TABLE B8

The Survival of Microbacterium in the Body (and Body Materials)

Factor(s)	Survival	Reference	
SKIN			
M. audouini			
Hair	420 d.	Farley	1921
Hair in well stoppered bottles	125 d.	Robinson	1948
Trichophyton interdigitale			
Scrapings from toes	300 d.	Mitchell	1922
SPUTUM			
Coccidioides immitis			
Sputum, sun with earth	Vegetative form, 30 d.	Rosenthal	1950
	Parasitic stage, 240 d.	Rosenthal	1950

TABLE B9

The Survival of Mycobacterium Species in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
M. tuberculosis			
Saline soln., R.T.	10 wks.	Coulthard	1951
In arachis oil	10 wks.	Coulthard	1951
In saline and arachis oil	At least 26 wks.	Coulthard	1951
Human whole blood	14 d.	Davies	1939
Hemoglobin inhibits the growth of the tubercle bacillus. Effects proportional to conc. of hemoglobin		Davies	1939
FECES			
M. tuberculosis			
Feces, R.T., dark,			
summer	1 d.	Abe	1949
fall	3 d.	Abe	1949
winter	9 d.	Abe	1949
Feces, 8 C.	Still virulent, 3½ mos.	Gärtner	1898
Dung	Virulent tubercle bacillus remained after 178 d.	Maddock	1933
Feces, No. of M.O. in feces proportional to those of sputums		Nüssel	1923
Feces, cool, stored in jars with loose lids, natural infected	12 mos.	Stenhause	1930
Feces, cellar, dark, with muslin to exclude insects artificially infected	2 yrs.	Stenhause	1930
Liquid manure	4 mos.	Stenhause	1930
Liquid manure, winter,			
artificially infected	5 mos.	Stenhause	1930
spring	2 mos.	Stenhause	1930
fall	4 mos.	Stenhause	1930
autumn	4½ mos.	Stenhause	1930
Cow feces, exposed on pasture-land, winter	5 mos.	Stenhause	1930
spring	2 mos.	Stenhause	1930
autumn	4 mos.	Stenhause	1930
summer	2 mos.	Stenhause	1930

FECES, (Continued)			
Cow feces, protected from sunlight, summer	4 mos.	Stenhause	1930
autumn	6 mos.	Stenhause	1930
M. paratuberculosis			
Infected feces, atmospheric conditions	246 d.	Lovell	1944
PUS			
M. tuberculosis			
Pus, R.T., dark	Viable 4 mos.	Abe	1949
Pus, R.T., in half dark	Viable 3 mos.	Abe	1949
SKIN			
M. tuberculosis			
Human tubercle bacillus in reed capsule placed under skin of cattle	7 yrs.	Heymans	1927
SPUTUM			
M. tuberculosis			
Sputum, R.T., dark	Viable 4 mos.	Abe	1949
Sputum, half dark	Viable 3 mos.	Abe	1949
Sputum, 37 C.	Persistence time 10 d., recov. time 0	Abe	1949
Sputum, direct sunlight			
summer	50–80 min.	Abe	1949
fall	60–110 min.	Abe	1949
winter	90–240 min.	Abe	1949
Sputum, diffuse light			
summer	8 d.	Abe	1949
fall	7 d.	Abe	1949
winter	5 d.	Abe	1949
On window of room			
spring	13 d.	Abe	1949
fall	18 d.	Abe	1949
summer	20 d.	Abe	1949
Sputum, exposed to sunlight, 12–14 C., R.H. 50%,			
afternoon	9 min., recov. 12+	Albini	1940
noon	1–4 min.	Albini	1940
diluted 1:2 (5:00 P.M.)	1 min., recov. 3+	Albini	1940
diluted 1:25	.....	Albini	1940
Sputum, exposed to sunlight, 7–9 C., R.H. 40%			
afternoon	1 min., recov. 3+	Albini	1940
diluted 1:2	4 min., recov. +	Albini	1940
diluted 1:25	.....	Albini	1940
Sputum, 43–35 C., R.H. 40%	.....	Albini	1940
Sputum, dried, surface of wooden tongue blade	Infective for G.P., 4–18 wks.	Arms	1912
Dried or moist	No viable after 3 yrs.	Burns	1917
Mixed with dust and exposed to sunlight up to 72 h.	Viable when inject. into G.P. 2–72 h.	Caldwell	1925
Droplets, 15–20 C.	30–40 d.	Chausse	1912
Droplets, 10–15 C.	50 d.	Chausse	1912
Direct sunlight	Few mins. to few h.	Cornet	1904
Diffuse sunlight	Several d.	Cornet	1904
Dried in layers not too thin	Several mos. to 1 yr.	Cornet	1904

SPUTUM, (Continued)			
Desiccated, 30–40 C.	9–10 mos.	De Thomas	1886
Sputum	6 mos.	Cornet	1904
Droplets from speaking or coughing, 45 C.	300–600 times increase in count	Flügge	1921
Loud speaking, droplets 45 C.	30 min.	Flügge	1921
Drops from bronchial tree	.....	Flügge	1921
Dried sputum	Several months	Harder	1913
Coughed on paper, stored in bell jar, dried 24 h.	>50% infect. in 2 d., none infect. in 31 d.	Kenwood	1915
M. tuberculosis			
Droplets	4–7 d.	Kirstein	1905
Desiccated	Several mos.	Koch	1882
Alternately dried and moistened	12 d.	Malassez	1883
Sunlight	Few minutes to 48 h.	Mayer	1924
Ultra-violet lamp for 3 min. at distance of 5″	Required 25 min. to destroy all organisms	Mayer	1924
Thin layer, dried	Still virulent, 30 min.	Migneco	1895
Direct sunlight	None recov., 24–30 h.	Migneco	1895
Dried in sun	18 h.	Migneco	1895
Exposed to light and air in June and July	<45 d.	Ransom	.....
Little or nor air and dark	16 d.	Ransom	.....
Dried 24 h., dark	>1 d.	Ransom	.....
Dried 24 h., dark, exposed to little air	>35 d.	Ransom	.....
Dried sputum	179 d.	Schill	1884
Desiccated	Several mos.	Schill	1884
Direct north roomlight, 45–76 F., R.H., 17–62%	4 h. to 5 d.	Smith	1942
Direct north roomlight, darkness	9 d. to 5 mos.	Smith	1942
45 F., R.H. 17–62%, dark	6½–14 mos.	Smith	1942
70 F., 83% R.H., dark, cover slip	142 d.	Smith	1942
63 F., 77% R.H., dark, water susp.	15 d.	Smith	1942
72 F., 79% R.H., dark	18 d.	Smith	1942
In India, direct sunlight	6–8 d.	Soparkar	1917
Desiccated in dark	309 d.	Soparkar	1917
Direct sunlight	6–8 h.	Soparkar	1917
Decomposing sput., test tube	20–26 d.	Soparkar	1917
Dried in thin smears	4 mos.	Sormani	1886
Dried sputum	10 mos.	Sormani	1886
Fluid sputum	8–11 d.	Toma	1886
Dark, moist box in paraffined bottles	Produce lesion in 170 d., no lesion in 188 d.	Twichell	1905
Dark closet	Lesion in 160 d., no lesion in 188 d.	Twichell	1905
Dark moist box, bottle stoppered with cotton	Lesion in 157 d., no lesion in 172 d.	Twichell	1905
Diffuse light, ordinary room, paraffined bottle	Lesion in 124 d., no Lesion in 175 d.	Twichell	1905
In ice	Lesion in 102 d., no lesion in 153 d.	Twichell	1905
Dark closet, bottles stoppered with cotton	Lesion in 100 d., no lesion in 141 d.	Twichell	1905
Moist, light place in sand	Lesion in 123 d., no lesion in 148 d.	Twichell	1905

SPUTUM, (Continued)			
M. tuberculosis			
Out of doors, winter mos., open bottles	Lesion in 110 d., no lesion in 132 d.	Twichell	1905
Handerchief	Lesion in 70 d., no lesion in 110 d.	Twichell	1905
Woolen blanket	Lesion in 70 d., no lesion in 110 d.	Twichell	1905
Wood	Lesion in 70 d., no lesion in 110 d.	Twichell	1905
In thermostat	Lesion in 33 d., no lesion in 70 d.	Twichell	1905
Dry light place, sand	Lesion in 30 d., no lesion in 70 d.	Twichell	1905
Carpet	Lesion in 39 d., no lesion in 70 d.	Twichell	1905
Direct sun	Lesion in 1 h., no lesion in 7 h.	Twichell	1905
Dried, R.T.	Infective 70 d.	Twichell	1906
Dried on carpet	Infective 39 d.	Twichell	1906
20 C., dark	4 mos.	Vidal	1934
2 C.	12 mos.	Vidal	1934
Dried	Several weeks	Villemin	1869
M. paratuberculosis			
Electric light	74–100 d.	Soparkar	1917
Direct sunlight	10–12 h.	Soparkar	1917
Diffuse light	30 d.	Soparkar	1917
Diffuse daylight	6–8 d.	Soparkar	1917
URINE			
M. tuberculosis			
Room temp., dark;	Viable 4 mos.	Abe	1949
in half dark	Viable 3 mos.	Abe	1949
2–3 C.	Viable 608 mos.	Abe	1949
60 C.	Recov. 0 in 1.5 h.	Nasta	1930
Refrigerator	Recov. 0 in 30 days	Nasta	1930
Inject into peritoneum of G.P.	Lose power in 30–40 d.	Nasta	1930
GENERAL			
M. tuberculosis			
Dried or broken up organic matter	Still virulent at 43 and 38 d., respectively	Cadeac Cadeac	1888 1888
Pieces of lung —1 to —8 C. exterior windowsill	<1 wk.		
Lung tissue inoc. and buried	167 d.	Cadeac	1888
Bacilli remain in phthisical cadavers for several years		Calmette	1920
Distilled water, 37 C.	<1 d.	Davies	1939
0.9% saline, 37 C.	<4 d.	Davies	1939
Lung of rabbit, .80 meter underground	>23 d.	Galtier	1887
In bile	Growth more rapid than in soap	Larson	1922
M. tuberculosis			
Rabbit tissue, buried in zinc box	3 mos., 6 d.	Petri	1921
Buried lung	2½ yrs.	Schottelius	1890
Intestinal contents, cow,			
not infect., autumn	4 mos.	Stillman	1938
not infect., summer	2 mos.	Stillman	1938

GENERAL, (Continued)			
Lung	3 yrs.	Stone	1891
Rabbit lung, liver, spleen kidney, 20 C., sterile, in vitro	5 d.	Vidal	1934
Rabbit tissue, 2 C.	10–30 d.	Vidal	1934
Lymph node emulsion in incubator in salt soln.	87 d.	Webb	1921
Lymph nodes coated with collodion	7 d.	Webb	1921
Liver and spleen coated with collodion	3 wks.	Webb	1921
Rabbit lung	14 mos. per rabbit following exposure to air	Wells	1941
Dead buried fowls	At least 1 yr.	Schalk	1928
M. paratuberculosis			
Intestinal scrapings mixed with river water, outdoor temp.	Recov. in 163 d.	Lovell	1944
B.C.G.			
Spleen of G.P.	53 d.	Gernz-Rieux	1950
Axillary lymph nodes	175 d.		

TABLE B10

The Survival of Micrococcus Species in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
M. spp.			
Sterile rabbit blood susp.	24 str. viable 9 yrs. 15 str. viable 19 yrs.	Frobisher	1947
FECES			
M. pyogenes var. aureus			
Feces, sunlight with heat	Bactericidal effect not marked	Lehmann	1931
SKIN			
M. pyogenes var. aureus			
Skin, 37 C., inoc. 161,000	Recov. 34,000 in 15 min.	Bryan	1933
Skin, left palm, expose m.o. 8 min., inoc. 2,568	Washed off 1,414	Burtenshaw	1938
Dead skin, expose 10 min. inoc. 2,568	Washed off 1,560	Burtenshaw	1938
Skin, inframammary fold,			
down	80% recov. in 1 h.	Cornbleet	1932
up	20% recov. in 1 h.	Cornbleet	1932
Skin, axilla, arm up	32% recov. in 30 min.	Cornbleet	1932
Skin, axilla, arm down	61% recov. in 30 min.	Cornbleet	1932
Interdig. spaces of toes,			
together	73% in 30 min.	Cornbleet	1932
apart	24% in 30 min.	Cornbleet	1932
Perianal fold, closed	76% recov. in 30 min.	Cornbleet	1932
open	28% recov. in 30 min.	Cornbleet	1932
Palms, clasped,	53% recov. in 30 min.	Cornbleet	1932
open	6% recov. in 30 min.	Cornbleet	1932
Growth in sweat of skin	Alkaline areas slower than acid in self-sterilizing action	Cornbleet	1932

SKIN, (Continued)			
Nail beds and areas surrounding nail beds	Less effective at sterilizing than normal areas	Cornbleet	1932
Keratotic areas	No more effective at removing *M. aureus* than normal areas	Cornbleet	1932
Moist areas	Depression of sterilizing power	Cornbleet	1932
Denuded epithelium	Not as effective in removing Staph. as intact areas	Cornbleet	1932
Previous exposure to U.V.	Does not enhance destructive power against Staph.	Cornbleet	1932
Skin of persons with furunculosis	Remain longer than in skin of others	Cornbleet	1932
Psoriatic lesions, bared of scales	Free themselves of Staph. faster than lesions with scales	Cornbleet	1932
Survived longer on skin than on filter paper		Hellot	1948
17.2% inhibited on normal skin		Hill	1933
M. pyogenes var. aureus			
Washed hide, inoc. 900	Recov. 360 in 30 min.	Norton	1932
Washed hide, inoc. 1,000	Recov. 25 in 20 min.	Norton	1932
Wounds	Recov. in 18 of 82 wounds	Rountree	1947
Dirty or fatty skin	Several hours	Singer	1929
SPUTUM			
M. pyogenes var. aureus			
Increased survival time dried sputum		Bordoni	1891
GENERAL			
M. pyogenes var. aureus			
Body temp.	Recovery 0	Arnold	1930
Pus, R.T.	2½–3½ yrs, none of pathogenicity lost	Bormann	1940
Exposed to long chain fatty acids	Lethal	Burtenshaw	1945
Pus, sunlight and heat	Bactercidal effect not marked	Lehmann	1931
Boils, 11 strains,			
pH 2.6	8 viable at 24 hrs.	Hall	1921
pH 5.0	3 viable at 24 hrs.	Hall	1921
pH 10.0	10 viable at 24 hrs.	Hall	1921
Boils, 11 strains,			
pH 2.6	10 viable at 7 days	Hall	1921
pH 5.0	1 viable at 7 days	Hall	1921
pH 8.0	1 viable at 7 days	Hall	1921
pH 10.0	10 viable at 7 days	Hall	1921
Abscesses, 4 strains,			
pH 2.6	2 viable at 24 hrs.	Hall	1921
pH 5.0	2 viable at 24 hrs.	Hall	1921
pH 8.0	0 viable at 24 hrs.	Hall	1921
pH 10.0	1 viable at 24 hrs.	Hall	1921
Abscesses, 4 strains,			
pH 2.6	2 viable at 7 days	Hall	1921
pH 5.0	2 viable at 7 days	Hall	1921

GENERAL, (Continued)			
pH 8.0	0 viable at 7 days	Hall	1921
pH 10.0	1 viable at 7 days	Hall	1921
Spinal fluid showed various pH ranges		Hall	1921
M. pyogenes var. albus			
Sputum, antrum nose, skin, throat, urine and uterus showed various pH ranges		Hall	1921
BLOOD			
Gaffkya spp.			
Sterile rabbit blood susp.	5 yrs.	Frobisher	1947
GENERAL			
Sarcina lutea			
Swabbed in large amounts on tongue, nasal mucous membranes, crypts of tonsils. Impossible to recover in very short time		Bloomfield	1919

TABLE B11

The Survival of the Neisseria Species in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
N. gonorrheae			
Sterile serum sealed with layer of paraffin	16 mos.	Ungerman	1918
Sterile serum sealed with layer of paraffin	7–8 wks.	Ungerman	1918
N. meningitidis			
Below 16 C., soaked in sterile horse serum	<24 hrs.	Downie	1940
Nasopharynx, body temp.	Avg. 6 mos. (longest 608 d.; shortest 34 d.)	Embleton	1919
N. spp.			
Sterile rabbit blood susp.	Neisseria—6 wks.–3 mos.	Frobischer	1947
URINE			
N. gonorrheae			
Male with urethritis	.....	Charles	...
20–26 C., pH 5–8.5	2 col. survived 48 hrs.	Carpenter	1933
Sediment 22 C., pH 5.0–8.5	8 col. survived 36 hrs.	Carpenter	1933
Sediment 4 C., pH 5.0–8.5	13 col. survived 36 hrs.	Carpenter	1933
pH increments of from 5.0–8.5	Strains isolated—8, 23, 17, 19, 15, 0, 2	Carpenter	1933
Temperature	.....	Allison	1942
GENERAL			
N. gonorrheae			
Urethral secretion, —5 to 2 C., pH 7.4–7.6, grown on ascitic agar;	1 hr. some reduction	Hamptman	1930
plus 12 C., pH 7.4	8 hrs. some reduction	Hamptman	1930
plus 22 C., pH 7.4	12 hrs. some reduction	Hamptman	1930

GENERAL, (Continued)			
Urethra, body conditions	<3 yrs.	Keyes	1911
Fresh pus, 22–23 C.	Still viable 24 hrs.	Schoñeld	1927
Cases successfully treated and operated on	No survival times	Cohn	1946
N. meningitidis			
Dried human secretions	Several days	Jungleblut	1935

TABLE B12

The Survival of Pasteurella Species in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
P. pestis			
Blood allowed to putrefy in test tube	100 d.	Ostertag	1908
P. of hemorrhagic septicemia			
Physiological serum	5 mos.	Iacotot	1926
FECES			
P. of swine plague			
Swine feces, 8 C.	Recov. 0 in 4–5 d.	Gartner	1898
P. spp.			
Sterile feces	Still alive 2 wks.–4 mos.	Gladin	1898
SPUTUM			
P. pestis			
Droplets	.....	Teague	1912
URINE			
P. pestis			
Sterile, R.T.	Still alive 3 mos.	Gladin	1898
GENERAL			
P. pestis			
Spleen of infected G.P., —15 C.	Virulent 14 mos., slightly vir. 2 yrs. 7 mos., dead 3 yrs. 5 mos.	Francis	1932
R.H. >37%, temp. >40 F.	Restricted to areas with these factors	Nohr	1951
Corpses of *C. pygmaeus*	Viable 23rd d. after death	Novikova	1934
Frozen cadavers in Manchuria	Viable 3 mos. after death	Strong	1942
Cadavers with putrid spleen	4 d.	Strong	1942
Cadavers after burial	3–30 d.	Strong	1942
Glycerine in spleen, —15 C.	Viable 7 yrs.	Frances	1932
P. tularensis			
23–26 C., internal organs	3 d.	Anon.	1947
Human tissue	Viable M.O. obtained from human tissue 2–3 mos. after pt. recov.	Foshay	1936
Spleens glycerinated, —14 C.	10–13 yrs.	Hull	1947
Frozen animal cord	42 mos.	Hull	1947
Frozen brain	36 mos.	Hull	1947
Frozen spleen	18 mos.	Hull	1947
Frozen muscle	12 mos.	Hull	1947
Frozen bone marrow	6 mos.	Hull	1947

TABLE B13

The Survival of Rickettsiae in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
R. conorii			
Human blood, ice box	12 d.	Blanc	1932
R. rickettsii			
Frozen brain, —70 C.	95 d.	King	1930
R. tsutsugamushi			
Mice blood and tissues	610 d.	Fox	1948
FECES			
R. andersoni			
Feces	6 yrs.	Philip	1948
TISSUES, GENERAL			
R. rickettsii			
—70 C., frozen brain	321 d.	King	1930
R. prowazeki			
Brain, 5 C.	79 d.	Macchiavello	1937
Tunica vaginalis, 5 C.	92 d.	Macchiavello	1937

TABLE B14

The Survival of Salmonella Species in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
Sterile rabbit blood susp.	7 yrs.	Frobisher	1947
FECES			
S. enteriditis			
Fluid stool, R.T., dark	8 d.	Dold	1944
Fluid stool, R.T.	8 d.	Dold	1943
Fluid stool, R.T., dark	11 d.	Dold	1944
Dried stool, dark, R.T.	53 d.	Dold	1944
S. paratyphi			
Original feces	74 d.	Dold	1947
Dried feces	421 d.	Dold	1947
Fluid stool, dark, R.T.	18 d.	Dold	1944
Stool dried on paper, R.T.	>8 mos.	Dold	1944
Fluid stool, dark, R.T.	20 d.	Dold	1944
Stool dried on paper, R.T.	>5 mos., 16 d.	Dold	1944
S. paratyphi B			
Fluid stool, R.T., dark	115 d.	Dold	1944
Fluid stool, R.T.	115 d.	Dold	1943
S. schottmülleri			
Fluid stool, R.T., dark	18 d.	Dold	1944
Stool dried on paper, R.T.	>8 mos.	Dold	1944
Fluid stool, dark	20 d.	Dold	1944
S. species			
Not found in feces of cockroaches	148 d.	Macfie	1922
Rat feces, R.T.		Welch	1941

FECES, (Continued)			
S. typhimurium			
Original feces	74 d.	Dold	1947
Dried feces	421 d.	Dold	1947
SKIN			
S. enteriditis			
Palmar surf., clean, R.T.	Recov. 0, 10 min.	Arnold	1930
Dirty hands, body temp.	5% gone, 20 min.	Arnold	1930
Hands, after washing	100% gone, 20 min.	Arnold	1930
Palm of hand	Destroyed in 10 min.	Krueger	1942
Dirty or fatty skin	Several hrs.	Singer	1929
S. paratyphi			
Dirty or fatty skin	Several hrs.	Singer	1929
S. typhimurium			
Turkey skin, frozen	13 mos.	Browne	1949
TISSUE, GENERAL			
S. typhimurium			
Chicken tissue, 93–96 C.			
after broiling	Inoc. 3.15×10^9/gm. Recov. .18% in 42 min.	Husseman	1951
after roasting	Inoc. 9.36×10^8/gm. Recov. 0.1% in 140 min.	Husseman	1951
BLOOD			
Normal goat serum, dried	Death rate logarithmic in response to conc. of surviving organisms	Chick	1912
Sterile rabbit blood susp.	7 yrs.	Frobisher	1947
Sterile rabbit blood susp.	1 str. viable 18 yrs.	Frobisher	1947
Blood and peritoneal fluid, R.T., air excluded	Virulence preserved >10 yrs.	Puntoni	1924
FECES			
Freezing	Several minutes	Beard	1940
Fluid stool, R.T., dark	4 d.	Dold	1944
Fluid stool, dark	4 d.	Dold	1943
Stool dried on filter paper, R.T.	>55 d.	Dold	1944
Stool, thin, watery, dark, R.T.	3 d.	Dold	1944
Stool, thin, watery, dried on filter paper, R.T.	>137 d.	Dold	1944
Feces in a latrine	30 d.	Galvagno	1908
Feces in a cask	25 d.	Galvagno	1908
Feces in earth of latrine, buried 10 d.	40 d.	Galvagno	1908
Feces in sand	5–20 d.	Galvagno	1908
Feces in loam	5–15 d.	Galvagno	1908
Feces in gravel	10–20 d.	Galvagno	1908
After being 10 days in latrine the organisms were no longer virulent		Galvagno	1908
8 C.	Recov. 0 in 5 d.	Gartner	1898
3 C.	Recov. 0 in 10 d.	Gartner	1898
Tap water with stool	Inoc. 100,000/cc water Recov.200/cc water in 2 d.	Horrocks	1911
R.T.	52 d.	Jordan	1926
Feces in latrine, winter	5 mos.	Levy	1903

FECES, (Continued)			
—8 to —30 F., exposed to open air	40–50 d.	Lu-ti-huan	1930
Not found in feces of cockroach		Macfie	1922
12–17 C.	Viable after 4–12 d.	Schiller	1890
Liquid manure	4 d.	Schiller	1890
Liquid manure, 13–22 C.	9 d.	Schiller	1890
Liquid manure, below 12 C.	8 d.	Schiller	1890
Liquid manure, above 8 C.	8 d.	Schiller	1890
Feces and urine, 16–20 C.	Viable 7 d.	Schiller	1890
8 d. cult., below 12 C.	Viable 7 d.	Schiller	1890
10 d. cult., below 12 C.	Viable 9 d.	Schiller	1890
13 d. cult., below 12 C.	Viable 27 d.	Schiller	1890
15 d. cult., 12–22 C.	Viable 9 d.	Schiller	1890
5 d. cult., below 12 C.	Viable 14 d.	Schiller	1890
5 d. cult., over 18 C.	Viable 8 d.	Schiller	1890
21 d. cult., below 12 C.	Viable 13 d.	Schiller	1890
Liquid manure, 12–17 C. 6 d. cult.	Viable 4 d.	Schiller	1890
Liquid manure, 10 d. cult.	Viable 4 d.	Schiller	1890
Liquid manure, 13 d. cult.	Viable 7 d.	Schiller	1890
Fresh feces, 17–22 C. weakly acid	Viable 115 d.	Uffelman	1889
Fresh feces, 17–22 C. neutral	Viable 121 d.	Uffelman	1889
Fresh feces and urine, —22 to 5 C., weakly alkaline	Viable 36 d.	Uffelman	1889
Old feces and urine, 17–22 C., alkaline	Viable 116 d.	Uffelman	1889
Old feces, 10 C., alkaline	Viable 116 d.	Uffelman	1889
Fresh feces, weak acid	Viable 44 d.	Uffelman	1889
Feces and urine in moist garden soil, 23–0 C.	Viable 5½ mos.	Uffelman	1889
Fresh feces and urine, neutral, 10 C. or less	Viable 66 d.	Uffelman	1889
Fresh feces and urine, weakly alkaline, 10 C. or less	Viable 21 d.	Uffelman	1889
Human feces on:			
dates	2 d.	Vasquez	1924
vinegar	1 h.	Vasquez	1924
mango	2 d.	Vasquez	1924
banana	2 d.	Vasquez	1924
apple	4 d.	Vasquez	1924
SKIN			
Self sterilizing ability of skin against organism due to drying		Dold	1919
Palm of hand	Entirely destroyed 10 min.	Krueger	1942
Washed hide	Inoc.550,Recov. 1, 30 min.	Norton	1932
Dirty or fatty skin	Several hours	Singer	1929
URINE			
Soaked in urine, dark	Inoc. 240,000, surv. 10 d.	Hewlett	1909
Soil and urine, dry, lab. temp.	Inoc. 12,000/gm soil, Recov. 330/gm. soil, 7 d.	Horrocks	1911
Garden humus with urine, exposed on veranda, no rain, dry	Inoc. 1,660/gm. soil, Recov. 280/gm. soil, 10 d.	Horrocks	1911
Warm temp.	<20–40 d.	Lu	1933

URINE, (Continued)			
—8 to —30 F., exposed to open air	40–50 d.	Lu-ti-huan	1930
12–17 C.	Viable after 4–12 d.	Schiller	1890
Urine infected water from well	A few days	Vacek	1932
7% urine, dil. ringers with 1% glucose	14 wks.	Zeller	1948
TISSUE, GENERAL			
Skin, clean	Inoc. 4,000, Recov. 1, 10 min.	Arnold	1930
Body temp., dirty fatty skin	Several hours	Arnold	1930
Palmar surf., clean, body temp.	Recov. 0, 10 min.	Arnold	1930
Human bile	>160 d.	Beckwith	1921
Beef bile	>141 d.	Beckwith	1921
Oyster juice and stomach, 50–60 F., ice broth cult.	30 d.	Foote	1895

TABLE B15

The Survival of Shigella Species in the Body (and Body Materials)

Factor(s)	Survival	Reference	
FECES			
S. ambigua			
Desert temp., in open box with flies	297 h.	Stewart	1944
S. dysenteriae			
R.T., 15–18 C.	From 5 to 15 d.	Cruickshank	1942
Fluid stool, R.T., dark	5–14 d.	Cruickshank	1940
Original feces	31 d.	Dold	1947
Dried feces	113 d.	Dold	1947
In artificially infected fecal material, the bacilli were detectable 3–4 d. when liquid and 33 d. dried		Dold	1943
(Ruhr bacillus), fluid stool	8.2 d.	Dold	1944
(Ruhr bacillus), dried stool	27.2 d.	Dold	1944
(Ruhr bacillus), fluid, R.T., dark	10 d.	Dold	1944
(Ruhr bacillus), dried in dark	1 mo.	Dold	1944
(Ruhr bacillus), fluid, dark, R.T.	28 d.	Dold	1944
Feces, 1.5–15 C., mixed with earth	>101 d.	Hampil	1932
Stool, cool, dried	1–2 wks.	Joe	1950
Fresh feces	5 d.	Kusama	1925
—8 to —30 F., exposed to open air	Up to 55 d.	Lu-ti-huan	1930
Not found in feces of cockroach		Macfie	1922
Desert temp., out of sun	11 d.	Stewart	1944
Human feces on mango	1 h.	Vasquez	1924
Human feces on banana	1 h.	Vasquez	1924
Human feces on apple	1 h.	Vasquez	1924
S. paradysenteriae			
40 C. (flexneri)	96 h.	Barnard	1946
20 C. (flexneri)	48 h.	Barnard	1946

Factor(s)	Survival	Reference	
FECES, (Continued)			
30 C. (flexneri)	24 h.	Barnard	1946
R.T. (flexneri)	Dried out quickly in fecal specimens	Cruickshank	1940
Dried feces (flexneri)	32 d.	Dold	1947
Oringal feces (Paradys.)	97 d.	Dold	1947
Dried feces (Paradys.)	270 d.	Dold	1947
Original feces (Hiss Y)	3 d.	Dold	1947
Dried feces (Hiss Y)	12 d.	Dold	1947
Original feces (flexneri)	31 d.	Dold	1947
Dried feces (flexneri)	113 d.	Dold	1947
Desert temp., out of sun	273 d.	Stewart	1944
Direct sun, all day	1 h. 20 min.	Stewart	1944
Human feces on mango	5 h.	Vasquez	1924
Human feces on banana	2 h.	Vasquez	1924
Human feces on apple	4 d.	Vasquez	1924
S. sonnei			
20 C.	72 h.	Barnard	1946
30 C.	24 h.	Barnard	1946
37 C.	6 h.	Barnard	1946
R.T.	6–11 d.	Cruickshank	1940
Feces	1–14 d.	Lowbury	1943
URINE			
S. dysenteriae			
Urine, warm, indoor	40–50 d.	Lu	1933
—8 to —30 F., exposed to open air	55 d.	Lu-ti-huan	1930
TISSUES, GENERAL			
S. dysenteriae			
Filtered human gastric juice, pH 4.5	Germicidal for Sonne-Duval strain of S. dysenteriae	Felsen	1939

TABLE B16

The Survival of Streptococcus Species in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
S. fecalis			
Sterile rabbit blood susp.	7–19 yrs.	Frobisher	1947
S. hemolyticus			
R.T., liquid paraffin, arachis oil	8 wks. 4 wks.	Coulthard	1951
Refrig., liquid paraffin, arachis oil	4 wks. >37 wks.	Coulthard	1951
Saliva, R.H., 40–55%	Mortality rates smaller than pneumococcus	Dunklin	1948
Sterile rabbit blood susp.	Alive 18 yrs. (Beta)	Frobisher	1947
Sterile rabbit blood susp.	Alive 9 yrs. (Alpha)	Frobisher	1947
S. liquefaciens			
Sterile rabbit blood susp.	7–19 yrs.	Frobisher	1947

BLOOD, (Continued)			
S. spp.			
Horse serum, 55 C.	.....	Belin	1933
37 C. for 3 hrs., defibrinated. Sprayed from chamber	Increased from 600 org. per unit to 50,000/unit in 1–7 days	Buchbinder	1941
SKIN			
S. pyogenes			
Fingers, exposed 2 min.	Inoc. 1,270, wash off 329	Burtenshaw	1938
Fingers, exposed 54 min.	Inoc. 1,228, wash off 190	Burtenshaw	1938
Fingers, exposed 118 min.	Inoc. 339, wash off 3.1	Burtenshaw	1938
Palm, exposed 2 min.	Inoc. 1,270, wash off 50.6	Burtenshaw	1938
Palm, exposed 54 min.	Inoc. 1,228, wash off .32	Burtenshaw	1938
Palm, exposed 118 min.	Inoc. 339, wash off 96	Burtenshaw	1938
Forearm, exposed 2 min.	Inoc. 1,971, wash off 157	Burtenshaw	1938
Forearm, exposed 64 min.	Inoc. 1,228, wash off 78	Burtenshaw	1938
Forearm, exposed 120 min.	Inoc. 339, wash off 78	Burtenshaw	1938
Dead skin, exposed 3 min.	Inoc. 1,971, wash off 88	Burtenshaw	1938
Dead skin, exposed 65 min.	Inoc. 1,228, wash off 232	Burtenshaw	1938
Dead skin, exposed 120 min.	Inoc. 339, wash off 14	Burtenshaw	1938
Left palm, exposed 4 min.	Inoc. 48, wash off .22	Burtenshaw	1938
Right palm, exposed 4 min.	Inoc. 48, wash off 0	Burtenshaw	1938
Exposed to long chain fatty acids	Lethal	Burtenshaw	1945*
S. hemolyticus			
Normal skin of hand, R.T.	Recov. 30 million, 3 min.	Colebrook	1930
From broth culture	Recov. 1.7 million, 1 h.	Colebrook	1930
	Recov. 7,000, 2 h.	Colebrook	1930
Hands of normal person	Group A isolated from hands of 7 out of 181	Colebrook	1930
	Non-hemolytic on nearly all of hands	Colebrook	1930
Skin of hands	6% viable, 1 h.	Krueger	1942
Washed hide	Inoc. 1,400, Recov. 400, 30 min.	Norton	1932
Surface, washed hide	Inoc. 1,000, Recov. 75, 30 min.	Norton	1932
Wounds	Alpha strep. found in 3 of 82 wounds	Norton	1932
S. pneumoniae			
Exposed to long chain fatty acids	Lethal	Burtenshaw	1945
S. viridans			
Exposed to long chain fatty acids	Lethal	Burtenshaw	1945
SPUTUM			
S. hemolyticus (Beta)			
Dried, 37 C.	Viable at 150 d.	Germano	1897
Saliva	In 20% no strep. demonstrable. 80% same strep. as in throat culture	Hamburger	1944
TISSUE, GENERAL			
S. hemolyticus (Beta)			
Dried membrane, 37 C.	Viable after 3 mos.	Germano	1897
Nasal secretion	Recoverable after last normal hand washing	Hamburger	1946

TISSUES, GENERAL, (Continued)			
Nose and throat, sulfadiazine 1 gm/day	More marked sensitivity in nose than throat	Hamburger	1946
Survived longer on skin than on filter paper.		Hellat	1948
Throat swabs, ice box, dried	½ yr.	Jettman	1927
S. spp.			
Animal excreta, 61 F.	Inoc. 1–3, Recov. .03 in 7 d.	Savage	1917

TABLE B17

The Survival of Vibrio Species in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
V. comma			
R.T., dry, glass smear, fowl	8 d.	Skidmore	1932
Turkey blood, 5 C., dried on glass	47 h.	Skidmore	1932
Turkey blood, 37.5 C., dried on glass	47 h.	Skidmore	1932
Beef bouillon, 37.5 C., in test tubes	Viable 5 wks.	Skidmore	1932
FECES			
V. comma			
Flies in contact with material rich in organisms	Found in feces	Alessandri	.....
Abs. humidity, 10–11 mg/cu. meter	Long enough for infection	Bey	1948
30 C.	Recov. 0 in 10 d.	Gartner	1898
8 C.	Recov. 0 in 5 d.	Gartner	1898
Rice water stools, dark cool weather	7–8 d.	Greig	1913
Rice water stools, dark warm weather	1–2 d.	Greig	1913
—8 to —30 F., exposed to open air	6–30 d.	Lu-ti-huan	1930
Feces	14 d.	Schiller	1890
Liquid manure	13 d.	Schiller	1890
Fresh feces and urine, 17–22 C., weak acid	Viable 24 h.	Uffelman	1889
9 C., weak acid	Viable 24 h.	Uffelman	1889
Old feces, thin with water,			
17–22 C., weak alkaline	Viable 96 h.	Uffelman	1889
9 C., weak alkaline	Viable 24 h.	Uffelman	1889
Fresh feces and urine, 17–22 C.	Viable 72 h.	Uffelman	1889
9 C. or less	Viable 24 h.	Uffelman	1889
Diarrheic feces and urine,			
17–22 C.	Viable 48 h.	Uffelman	1889
9 C. or less	Viable 48 h.	Uffelman	1889
Human feces on chico, cucumbers, papaya, lettuce	Overnight	Vasquez	1924
Human feces on cut cucumbers	>20 h.	Vasquez	1924
Human feces on cut papaya	<44 h.	Vasquez	1924
Human feces on shrimp	22 h.	Vasquez	1924

FECES, (Continued)			
Human feces on oysters	46 h.	Vasquez	1924
Human feces on inside open clam	4 d.	Vasquez	1924
Human feces on dates	2 h.	Vasquez	1924
Human feces on vinegar	1 h.	Vasquez	1924
Human feces on mango	2 d.	Vasquez	1924
Human feces on banana	2 d.	Vasquez	1924
URINE			
V. comma			
Urine, warm, indoor	<20–40 d.	Lu	1933
—80 to —30 F., open air	6–30 d.	Lu-ti-huan	1930
Urine	14 d.	Schiller	1890

TABLE B18

The Survival of Viruses in the Body (and Body Materials)

Factor(s)	Survival	Reference	
BLOOD			
Hoof and Mouth virus			
—20 C., thawed at R.T., 24 h. previous to test	6–7 wks.	Henderson	1948
—10 to —20 C., thawed when tested	5¾ mos.	Henderson	1948
G.P. lymph, pH 7.5, in phosphate buffer	2 yrs. and 20 d.	Sichert-Modrow	1930
Dried lymph 70 C., dried in P_2O_5 in vacuo	2½ h.	Sichert-Modrow	1930
Dried lymph 122 C., dried with P_2O_5 in vacuo	3 min.	Sichert-Modrow	1930
Dried lymph of G.P., 52 C. in P_2O_5	Still infectious 14 h.	Sichert-Modrow	1930
Dried lymph of G.P., R.T.	Still infectious 10 d.	Sichert-Modrow	1930
Dried serum, R.T.	Still infectious 5 d.	Sichert-Modrow	1930
Dried plasma, R.T.	Still infectious 7 d.	Sichert-Modrow	1930
Dried preserved serum, R.T.	Still infectious 48 d.	Sichert-Modrow	1930
Fresh G.P. lymph, 2–7 C., stored in cold room	Retained virulence 190 d.	Stockman	1926
Herpes			
U.V. radiation, fresh normal rabbit serum	10 min.	Gunderson	1932
Normal rabbit serum	40 min.	McKinley	1926
Mexican typhus			
Blood and tissue, 5 C.	79 d.	Macchiavello	1937
Yellow Fever virus			
Blood dried in vacuum while frozen	154 d.	Sawyer	1929
Monkey blood with liver, —10 C., in sealed sterile test tube	2 wks.	Sellards	1928

BLOOD, (Continued)			
Rift Valley Fever			
Blood, refrigerator	82 d.	Smithburn	1949
Blood, refrigerator (stored, fluid)	2 yrs.	Smithburn	1949
Serum, refrigerator	1,048 d.	Smithburn	1949
FECES			
Hoof and Mouth virus			
Inoc. G.P. tissue and stored —20 C.	2–3 mos.	Andrews	1931
Moist, cool, 3.5–5.5 C., for 105 d., dried rapidly in vacuum, R.T.	283–345 d.	Hull	1947
Infective Jaundice			
Dust borne, dried	31 d.	Anderson	1947
Newcastle virus			
Dried chicken feces, pH 6.3 and 6.8		Olesiuk	1951
Psittacosis			
Exposed to HCOH, 0.2%, R.T.	Infective up to 10 d.	Hull	1947
Polio virus			
3–4 C.	>6 mos.	Kramer	1940
Stools	7–8 wks.	Horstmann	1944
Feces of child	Recov. 7 d., after onset of illness	Kramer	1939
Stools, virus found in stools, inactivated by inactivated sludge and chlorination		Krumbiegel	1944
Stools of convalescents	25 and 123 d. after mild abortive polio	Lepine	1939
Feces of polio victims and carriers	Possibly a matter of hrs.	Maxcy	1943
Stool	<6% viable	Paul	1941
Stool in transatlantic mail	16 d.	Paul	1939
Aqueous stool susp., ice box	2½ mos.	Paul	1940
Refrigerated	10 wks.	Trask	1938
Stools of convalescents	25–123 d. after abortive cases	Trask	1938
Refrigerated	75 d.	Trask	1938
SKIN			
Influenza virus			
Human skin, inoc. 0.2 cc. virus susp.	50% recov. in 10 min.	Krueger	1942
Suspended in chick allantoic fluid, skin of hand	45–50 min.	Parker	1944
Vaccinia virus			
Light reduced ½	Pustule up to 8 h.	Herzberg	1933
SPUTUM			
Virus spp.			
R.T.	Recov. positive, 92 d.	Arloing	1927
GENERAL			
Hoof and Mouth virus			
Inoc. G.P., 2 C.	2–3 mos.	Andrews	1931

GENERAL, (Continued)			
Beef, —20 C., buffered sol.	4 mos.	Henderson	1948
Tissues, liver, kidney, rumen, 4 C.	24 h.	Henderson	1948
Liver, rumen, lymph node, —10 to —20 C., thawed when tested	5¾ mos.	Henderson	1948
4 C., pH 5.6	<3 d.	Henderson	1948
—20 C., pH 6.8, thawed in buffered sol'n.	11 d.	Henderson	1948
120 C., pH 6.6, thawed in buffered sol'n.	4 mos.	Henderson	1948
Muscles, near 0	1 wk.	Rubino	1929
Bone	At least 40 d.	Rubino	1929
Carcass, —20 C., pH 6.8 thawed at R.T.	4 mos.	Henderson	1948
Rinderpest virus			
Live rabbits and storage	7 d.	Haddow	1947
Polio virus			
Brain tissue, 37 C., in ascitic fluid kidney	20 but not 30 d.	Flexner	1915
Polio stored in animal tissue over long period of time does not retain viability		Flexner	1917
Nasal washings of child	Recov. 5 d. after onset of illness	Kramer	1939
Nasopharyngeal washing	Recov. 9 d. after onset of illness	Kramer	1939
Pharynx	Recov. 10% viable	Paul	1941
Fresh prep. in2amoeba proteus	<3 d.	Toomey	1948
Rabies			
Rabbit brain, 5 C.	>68 d.	Remlinger	1934
Brain in glycerin, in disintegrator	47 d.	Barrat	1904
Brain susp. in liquid air, dil. 1:10	24 h.	Barrat	1904
pH 2	1 h.	Dresel	1934
Acids and alkalis harmful in proportion to conc.		Dresel	1934
Herpes virus			
20% susp. of rabbit brain in buffered physiological saline, 37 C.	100 h.	Book	1940
Brain emulsion	40 min.	McKinley	1926
U.V. radiation, cornea	<15 min.	Gunderson	1932
Yellow Fever virus			
Liver, —12 C.	>1 mo.	Sawyer	1929
Mouse brain, 8 C.	Non-virulent, 160 d.	Theiler	1930
Mouse brain plus 50% glycerin, stored at 2–4 C.	Infective after 58 d. but not often 100 d.	Theiler	1930
Mouse brain in whole monkey serum, —78 C.	6 mos.	Turner	1938
Influenza virus			
Palmar skin	Recov. 0 in 10 min.	Anon.	1943
Mouse brain susp. in N rabbit serum, —20 to —30 C.	<6 mos.	Olitsky	1949
Mouse lung susp. in 10% plain broth, —78 C.	6 mos.	Turner	1938
Frozen rabbit testes, —78 C.	3 yrs.	Turner	1939

GENERAL, (Continued)			
Newcastle Disease virus			
Skin, eviscerated carcass, 34 F., exp. infected	96 d.	Asplin	1949
Bone marrow	134 d.	Asplin	1949
Unplucked carcasses, skin bone marrow	160 d. 196 d.	Asplin Asplin	1949 1949
Fowlpox virus			
Dried material from lesions	2 yrs.	Loewenthal	1906
Vaccinia virus			
Mouse brain, 4 C., dried over $CaCl_2$ in sealed glass vessels	Still virulent to mice in 71 h.	Haagen	1939
Mouse brain tissue, 20% glycerin in water, strain II Neurolapine R.G.A.	Recov. after 28th passage 10 mos.	Haagen	1939
Mouse brain, temp. of freezing-drying apparatus, strain II Neurolapin R.G.A.	Recov. after 1 yr. 9 mos.	Haagen	1939
Mouse brain tissue, 4 C., strain II L 3 b.	Recov. after 2 yrs.	Haagen	1939
Mouse brain tissue plus glycerin, refrigerated, strain II L 3 b.	Not virulent after 1 yr.	Haagen	1939
Mouse brain, temp. of freezing-drying apparatus, strain II L 3 b.	Still showed skin reaction after 1 yr.	Haagen	1939
Mouse brain plus glycerin strain II L 3 b.	Still showed skin reaction after 6 mos.	Haagen	1939
Chorion plus calf lymph, temp. of refrigerator, strain III Breslan	Still showed skin reaction after 2 yrs.	Haagen	1939
Chorion plus calf lymph, temp. of refrigerator, strain IV Dresden	Still virulent after 2 yrs.	Haagen	1939
Chorion, 4 C. strain V Hanover	Not given	Haagen	1939
Neurotropic viruses			
—20 to —30 C.	9 mos.	Olitsky	1949
Encephalitides			
Brain tissue, 50% glycerin	1 yr.	Hull	1947
Dried	Lose virulence	Hull	1947
Frozen	>3 mos.	Hull	1947
pH below 5.5	Lose viability	Hull	1947
pH 9.2	Viability returns	Hull	1947
Mouse brain susp. in N rabbit serum —20 to —30 C. (Jap. B encephalitis)	<6 mos.	Olitsky	1949
Mouse brain emulsion in plain broth, —78 C.	6 mos.	Turner	1938
Meningo-meningitis			
Frozen rabbit testes, —78 C.	3 yrs.	Turner	1939
Lymphogranuloma inguinale			
Frozen rabbit testes, —78 C.	10 mos.	Turner	1939
Smallpox virus (Variola)			
Dry crusts, R.T., daylight and dark	Long periods >1 yr.	Downie	1947
Pneumoenteritis virus			
Calf lung, 60, filtered thru Berk. or Seitz filter	Recov. 0 in 10 min.	Gallo	1948

GENERAL, (Continued)			
55 C., filtered, dried	Still active in 10 min.	Gallo	1948
Dried lung in refrigerator	No activity, 20 d.	Gallo	1948
Frozen dried lung	No. activity, 6 d.	Gallo	1948
Virus spp.			
Some viruses retain their activity in vitro for several years. Some bacteriophage endure for a long time in bacteria free filtrate		Boycott	1928
Leukocytes and spleen tissue, 4 C., dried by ppt. by cold acetone	>2 mos.	Das	1949
4 C., dried over calcium chloride in vacuum	>4 mos.	Das	1949
37 C.	15 d.	Das	1949

TABLE B19

The Survival of General Bacteria in the Body (and Body Materials)

Factor(s)	Survival	Reference	
Gastric juice	Organisms recov. 5½ hrs. after feeding dogs 500 cc. alk. milk with organisms	Arnold	1926
Sweat. Freshly secreted. May have mild antiseptic value due to acid pH imparted by lactic acid		Bergeim	1943
Tissue surfaces in contact with environment rich in bacteria as respiratory and digestive tract have strong inhibitory effect on bacteria which is lacking on tissue surfaces of the body cavities such as pleura, pericardium, peritoneum. Saliva plays important role since it contains inhibitors sensitive to cold, soluble in water and precipitated by alcohol, chloroform, and acetone		Dold	1947
4 d. since bath. Skin scrapings	Deltoid area: aerobic, 70 organisms anaerobic, 37 organisms	Dold	1947
7 d. since bath. Skin scrapings	Scapular area: aerobic, 47 organisms anaerobic, 10 organisms	Evans	1950
Saliva sprayed in air	36 hrs.	Lukriech	1947
Hands and arms	Inoc. 4,580,000, recov. 1,000,000 after scrubbing 14 min.	Price	1938
Sores on man and beast, 110–120 F. Sun on sores	Organisms destroyed	Sternberg	1894

SUMMARY OF ABBREVIATIONS USED IN TABLES

alk.	alkaline	org.	organism
avg.	average	path.	pathogenic
C.	Degrees centigrade	physiol.	physiological
Col.	Colonies	ppm.	parts per million
conc.	concentration	ppt.	precipitate
cont'd, cont.	continued	R.H.	Relative humidity
ct.	count	R.T.	Room temperature
cult.	culture	Recov.	Recovered
d., ds., das.	day or days	refrig.	refrigeration
Dessic.	Desiccate	sec.	second
dil.	dilution	sensit.	sensitization
F.	Degrees fahrenheit	soln., sol'n.	solution
fl.	fluid	spp.	species
G.P.	Guinea pig	str.	strain
gel.	Gelatin	susp., susp'n.	suspension
h., hrs.	hour or hours	T.B., tb.	tuberculosis
inc.	increase	temp.	temperature
Inoc., Innoc.	Inoculate	U.V., U.v., UV	Ultra violet
irrad.	irradiated	wks.	weeks
Lg.	Large	×	times
max.	maximum	yr., yrs.	year or years
med.	medium	>	greater than
met.	methyl	<	less than
min.	minute or minutes	+	present; plus
mos.	months	0	none
mult.	multiplied	—	minus

REFERENCES

Abe, T.: Ueber die Lebensdauer der Tuberkelbazillen in Exkreten. I. Einflub der Temperatur. *Tohoku J. Exper. Med.*, 50: 329–336, 1949.

Abel, R.: Beitrage zur Frage der Lebensdaner die Diphtheriebacillen. *Centr. f. Bakt.*, 14: 756–761, 1893.

Abel, R.: Zur Kenntnis des Pestbacillus. *Centr. Bakt.*, 21: 497–517, 1897.

Albini, A.: Action of sunlight and of other meterologic factors on tubercle bacilli. *Rassegna internaz. di Clin. E Terap.*, 21: 828–833, 1940.

Alessandrini, G., and G. Sampietro: The vitality of cholera vibriones in milk and on the fly. *Ann. Igiene Sper.*, 22: 623–650, 1912.

Allison, S. D., R. Charles, and C. M. Carpenter: Survival time of gonococcus in urine from male patients with urethritis. *Venereal Disease Inform.*, 23: 283–286, 1942.

Amoss, H. L., and M. A. Poston: Undulant (Malta) fever: isolation of the brucella organism from the stools. *J. Am. Med. Assoc.*, 93: 170–171, 1929.

Anderson, I. S.: Infective jaundice in Iceland. *Lancet*, 252: 778–781, 1947.

Andrews, W. H., N. Dobson, T. Bannatyne, G. O. Davies, G. B. Simmins, C. V. Watkins, and J. T. Evans: *Foot and Mouth Disease Commission*, London, 1931.

Anonymous: *Thirty-third annual report. Agric. Exptl. Station, La.* 1921.

Anonymous: *Official history of the war Medical Services. Pathology*, London, 142, 1923.

Anonymous: *Rep. N.Y. St. Vet. Coll.* 1931–1932, 1933.

Anonymous: The inactivation by human skin of influenza virus in the presence of saliva. *U.S. Naval Med. Bull.*, 41: 692–697, 1943.

Arguelles, M. V.: Viability of cholera vibrio in various menstrua. *Rev. Filipina de Med. y Farm.*, 18: 137–140, 1927.

Arkharow, J.: Recherches sur la guerison de l'infection pneumonique chez les lapins au moyen du serum des lapins vaccines. *Arch. d. med. exper. et d'anat. path.* 4: 498, 1892.

Arkwright, J. A., and A. W. Balot: Investigation of the etiology of typhus fever, especially undertaken for the Egyptian government in the Public Health Laboratries, Cairo. *Brit. J. Exp. Path.*, 4: 70, 1923.

Arloing, F., and A. Dufourt: Temps de conservation du Virus filtrant tuberculeaux dans les produits pathologiques. *Compt. rend. soc. biol.*, 96: 453–455, 1927.

Arms, B. L., and E. M. Wade: Variations in the longevity of tubercle bacilli from different individuals. *Trans. Nat. Assoc. Prev. Tuberc.*, 493– 497, 1912.

Armuzzi, G., and R. Strempel: On the behaviour of Spirochaeta pallida in dead tissue. *Arch. F. Dermat. u. Syph.*, 150: 295, 1926.

Arnold, L., and A. Bart: The self-disinfecting power of the skin. *Am. J. Hyg.*, 19: 217–228, 1934.

Arnold, L., and L. Brody: The gastro-duodenal bactericidal mechanism. *Am. J. Hyg.*, 6: 672, 1926.

Arnold, L., C. J. Gustafson, T. G. Hull, B. E. Montgomery, and C. Singer: The self-disinfecting power of the skin as a defense against microbic invasion. *Am. J. Hyg.*, 11: 345, 1930.

Asplin, F. D.: Observations on the viability of Newcastle disease virus. *Vet. Rec.*, 61: 159–160, 1949.

Atkinson, N.: The occurrence of Aerobacter aerogenes in sewage and feces. *Australian J. Exp. Biol. Med. Sci.*, 12: 141–149, 1934.

Bang, B.: Die Aetologie des seuchenhaften Verwerfens. *Z. f. Thier.*, 1: 241–278, 1897.

Barnard, H. F.: Effect of temperature on survival of *Shigella sonnei* and *Shigella flexneri* in feces. *Month. Bull. Min. Health and Emerg. Pub. Health Lab. Serv.*, 5: 261–264, 1946.

Barrat, J. O. W.: Contrifugalisation and Disintegration in relation to virus of rabies. *Centr. Bakt.*, 35: 769–795, 1904.

Beard, P. J.: Longevity of Eberthella typhosus in various soils. *Am. J. Pub. Health*, 30: 1077, 1940.

Beaver, J. C., and G. Deschamps: The viability of *E. histolytica* cysts in soil. *Am. J. Trop. Med.*, 29: 189–191, 1949.

Beck, Dorothy M.: California field and laboratory studies on relapsing fever. *J. Infectious Diseases*, 60: 64–80, 1937.

Beckwith, T. D., and R. H. Lyons: Viability and growth of *B. typhosus* in bile. *J. Infectious Diseases*, 28, 62–66, 1921.

Belin, M., S. Mutermilch, and E. Salmon: Effect of protective substances on heat resistance of certain bacteria. *Compt. Rend. Soc. de Biol.*, 164: 1278–1281, 1933.

Bergeim, O., and T. Cornbleet: Antibacterial action of lactic acid and volatile fatty acids of sweat. *Am. J. Med. Sci.*, 205: 785–792, 1943.

Bey, M. K.: Effect of absolute humidity of atmosphere on first wave of cholera epidemic in Egypt in 1947. *Jour. Roy. Egyptian M.A.* 31: 39–72, 1948.

Blair, J., and O. Lang: Effect of low temperature freezing on the encysted larvae of *Trichlinella spiralis*. Studies on muscle of rats, guinea pigs and hogs. *Jour. Infect. Dis.* 55: 95–104, 1934.

Blanc, G., and M. Baltazard: Long period of conservation, in dried excrement of infected fleas, of typhus virus. *Compt. rend. Acad. de Sci.*, 204: 1046–1048, 1937.

Blanc, G., and M. Baltazard: Longevity of virus of murine typhus in excrements of infected fleas. *Bull. Soc. Path. Exot.*, 33: 25–32, 1940.

Blanc, G., and J. Caminopetros: Etudes epidemiologiques et experimentales sur la fievre boutonneuse faites a L'institut Pastuer d'Athenes. Arch. Inst. Pasteur, Tunis, 20: 343–349, 1932.

Bloch, O., Jr.: Loss of virulence of Treponema pallidum in citrated blood at 50° C. *Bull. Johns Hopkins Hosp.*, 68: 412–415, 1941.

Bloomfield, A. L.: Fate of bacteria introduced into upper air passages. *Bull. Johns Hopkins Hosp.*, 30: 317, 1919.

Boak, R. A., C. M. Carpenter, and S. L. Warren: Thermal death time of *Treponema* pallidum in vitro, with special reference to fever temperatures. *J. Exp. Med.*, 56: 741–750, 1932.

Bordoni-Uffreduzzi, G.: Sulla Resistenzo Del Virus Phneumonico Nezli Sputi. *Arch. Sci. Med.*, 15: 341–348, 1891.

Bormann, F. von: Beitrag uber die Haltbarkeit der Staphylococcen im Eiter. *Zentr. Bakt.*, 146: 68–69, 1940.

Boycott, A. E.: Ankylostomiasis. *Trans. Epidemiological Soc.*, London, 24: 113–142, 1905.

Braun, W., A. N. Gorelick, M. Kraft, and D. Mead: Studies on the in vivo fate of brucella variants with different virulence. *J. Infectious Diseases*, 89: 286–295, 1951.

Brown, H. W.: Studies on the rate of development and viability of the eggs of *Ascaris lumbricoides* and *Trichuris trichiura* under field conditions. *J. Parasitol.*, 14: 1–15, 1927.

Browne, A. S.: *The public health significance of salmonella on poultry and poultry products.* Ph.D. Thesis, U. Calif., 1949.

Brumpt, E.: Longevite du virus de la fievre boutonneuse chez la tique, *Rhipicephalus sanguinens. Compt. rend. soc. de biol.*, 110: 1199–1202, 1932.

Bryan, C. S. and W. L. Mallman: Some factors responsible for the so-called self-disinfecting power of the skin. *J. Lab. Clin. Med.*, 18: 1249–1255, 1933.

Buchanan, G.: *Spec. Rep. Ser. Med. Res. Coun.*, London, 113, 1927.

Buchbinder, L., M. Solotorovsky, M. Solowey, and J. Ruhlkoupal: Studies on microorganisms in simulated room environments. V. The effect of survival on the pathogenic properties of streptococci: properties other than mouse virulence. *J. Bact.*, 42: 631–634, 1927.

Burke, G. S.: Occurrence of B. botulinus in nature. *J. Bact.*, 4: 541, 1919.

Burns, N. B.: Some experiments with tuberculous sputum twenty-two years old. *Am. Rev. Tuberc.*, 1: 484–488, 1917.

Burtenshaw, J. M. L.: Mortality of Streptococcus hemolyticus on skin and surfaces. *J. Hyg.*, 38: 575–586, 1938.

Burtenshaw, J. M.: Self-disinfection of the skin: a short review and some original observations. *Brit. Med. Bull.*, 3: 161–164, 1945.

Cadeac, N. I., and N. I. Malet: Research experiments on the virulence of purulent matter, dried, putrified or frozen. *Congres pour l'etude de la tuberculose*, 130: 1888.

Caldwell, M. E.: Viability of Mycobacterium tuberculosis in semi-arid environments. *J. Infectious Diseases*, 37: 465–472, 1925.

Caldwell, F. C., and E. L. Caldwell: Preliminary report on observations on the development of ova of pig and human Ascaris under natural conditions, and studies of factors influencing development. *J. Parasitol.*, 14: 254, 1928.

Calmette, N. I.: L'infection bacillaire et la tuberculose. 1920.

Cameron, H. S.: *Rep. N.Y. St. Vet. Coll*, 1933, 1931–1932.

Canfora, M.: Ueber die Latenz der Tetanussporen im tierischen Organismus. *Centr. Bakt., Abt. I., Orig.*, 45: 495–501, 1907.

Cedar, E., R. Dyer, A. Rumreich, and L. Badger: Typhus fever; typhus virus infeces of infected fleas (Xenopsylla cheopis) and duration of infectivity of fleas. *Pub. Health Rep.*, 46: 3103–3106, 1931.

Chick, H.: The bactericidal properties of blood serum. *Jour. Hyg.*, 12: 414–436, 1912.

Chief, Military, Sanitary Admin. of the Red Army. *Manuel of Infectious Diseases Medgiz*, Moscow, 201, 1944.

Cohn, A., L. Grunstein, and C. E. Heaton: Persistence of gonococcus in female adnexa. *Am. J. Obstet. and Gynecol.*, 52: 83–88, 1946.

Cohn, E. G.: Koliforme bakterier i kogdning og ra maelk. *Nordisk Veterinaermed*, 2: 209–236, 1950.

Colebrook, L.: *Min. Hlth. Inter. Rep. Dep. Comm. Matern. Mortal. Morb.*, Appendix D. 1930.

Colebrook, L., W. R. Maxted, and A. M. Johns: Presence of hemolytic and other streptococci on human skin. *J. Path., Bact.*, 41: 521–527, 1935.

Cornbleet, T.: Self-sterilizing powers of the skin. II. *Arch. Dermatol, and Syphilol.*, 25: 1058–1059, 1932.

Cornbleet, T.: Self-sterilizing powers of the skin. V. *Arch Dermatol, and Syphilol.*, 28: 526–531, 1934.

Cornbleet, T., B. E. Montgomery: Self-sterilizing powers of the skin. I. *Arch. Dermatol. and Syphilol.*, 23: 908–919, 1931.

Cornet, G.: *Tuberculosis and acute generalized tuberculosis.* American edition by James, W. B., and A. Stengel. 1904.

Cornet, G.: *Tuberculosis*, 83: 1904.

Cort, W. W.: Investigations on the control of hookworm disease. XXXIV. *Am. J. Hyg.*, 5: 49, 1925.

Cort, W. W., J. B. Grant, N. R. Stoll, *et al.*: An epidemiologic study of hookworm disease in the mulberry districts of the Yangtze delta. *Am. J. Hyg.*, Monog. No. 7: 1926.

Coulthard, C. E., B. H. Chantrell, and B. Croshaw: The survival of streptococci and tubercle bacilli in oils; with special reference to those of pharmaceutical interest. *J. Pharmacol.*, London, 3: 4, 215–226, 1951.

Cruickshank, R., and M. Gillespie: Diagnosis of intestinal infections. *Lancet*, 2: 322, 1942.

Cruickshank, R., and R. Swyer: Outbreak of Sonne dysentery. *Lancet*, 803, 1940.

Da Rocha-Lima, H.: Zur Aetiologie des Fleckfiebers. *Berliner Klin. Wochenschrift*, 53: 567–569, 1916.

Das, N. B.: Studies on the drying of rinderpest virus. *Indian Jour. Vet. Sci. and Animal Husbandry*, 19: 5–10, 1949.

Davies, R.: Influence of normal blood and certain of its constituents on growth and viability of the tubercle bacillus. *Tubercle*, 20: 5–49, 1939.

Davis, G. E.: Further attempts to transmit *Pasteurella tularensis* by the bedbug. *J. Parasitol.*, 29: 395–396, 1943.

De Thoma, N. I.: Sulla Virulenza Dello Sputo Tuberculare. *Annali. Univers. Di Medi Juillet.* 1886.

D'Herelle, F., R. H. Malone, and M. N. Lahiri: Studies on Asiatic cholera. *Indian Med. Res. Mem.*, No. 14, 1930.

Dobell, C. E.: Researches on the intestinal protozoa of monkey and man. VIII. An experimental study of some Simian strains of "*Endamoeba coli.*" *J. Parasitol.*, 28: 541–593, 1936.

Dold, H.: Ueber die Infektionsgefahr durch Wasche, die mit Krankheitskeimen (Typhus, Paratyphus, Enteritis und Ruhrbasillen) verunreinigt ist. *Arch. Hyg. u. Bakt.*, 131: 134–149, 1943.

Dold, H.: Danger of infection from "washing" soiled with organisms of typhoid-paratyphoid-dysentery group. *Bull. War Med.*, 5: 132, 1944.

Dold, H.: Keim-inhibition (Kelmvermehrungshemmung) als Mittel der Mikrobienabwehr., *Med. Klin.*, 42: 45–48, 1947.

Dold, H., Chen Yu Hsiang: Uber das Verhaltinis der tatsachilichen zur theoretisch Moglichen gefahr der Keimubertragung durch Fingerberuhrungen. *Z. Hyg. Infektionskrankh.*, 89: 266–284, 1919.

Dold, H., and M. Ketterer: Vergleichende Untersuchungen uber die Wachweisbarkeit von Ruhrbazillen in flussigem und getrocknetem Stuhlmaterial. *Z. Hyg. u. Infektionskrankh.*, 125: 215–225, 1943.

Dold, H., and M. Ketterer: Comparative studies on survival time of bacteria of typhoid-paratyphoid-enteritis group and dysentery group in liquid fecal matter and in feces dried on filter paper. *Z. Hyg. u. Infektionskrankh,*. 125: 444–456, 1944.

Dold, H., and M. Ketterer: Weitere vergleichende Untersuchungen uber die Nach weisbarkeit von Bakterien der Ruhrgruppe und der T.P.E. gruppe in flussigem oder an filtrier Papier angtrocknetem Stuhlmaterial. *Z. Hyg. Infektionskrankh.*, 127: 326–333, 1947.

Downie, A. W.: Survival of meiningococci on swabs and blood. *Lancet*, 2: 36–37, 1940.

Downie, A. W., and K. R. Dumbell: Survival of *Variola* virus in dried exudate and crusts from smallpox patients. *Lancet*, : 550–553, 1947.

Dresel, E. G., and F. Sander: Distribution of rabies virus in the organs of rabbits and the influence of acids and alkalis on the virus. *Z. Immunitatsforsch.*, 81: 457, 1943.

Du Buy, H., F. A. Arnold, and B. J. Olson: Air transmision of microorganisms derived from the respiratory tract. *I. Lactobacillus acidophilus* as a test organism. *Pub. Health Repts.*, 62: 1391–1413, 1947.

Duguid, J. P.: Expulsion of pathogenic organisms from the respiratory tract. *Brit. Med. J.*, 1: 265, 1946.

Dunklin, E. W., and T. T. Puck: The lethal effect of relative humidity on airborne bacteria. *J. Exptl. Med.*, 87: 87–100, 1948.

Dykstra, R., L. Roderick, H. Farley, and V. McMahan, and E. Splitter: *Tech. Bull. Kansas Agric. Exptl. Sta.* 66: 1–24, 1948.

Easton, E. J., and K. F. Meyer: Occurrence of Bacillus botulinus in human and human and animal excreta. *J. Infectious Diseases*, 35: 207–212, 1924.

Eichorn, H.: *Beitrage zum Kenntniss des Einflussen vom temperature und Witterungsverhaltnissen auf die tenacitat des Tuberkulosen Sputums.* Jena, B. Eugan., 1893.

Embleton, D., and G. H. Steven: The persistence of cerebro-spinal fever cases as carriers. *Lancet*, 1: 788–790, 1919.

Emmerich, R.: Ueber die Infection, Immunisirung, und Heilung bei crouposer Pneumonie. *Z. Hyg. u. Infektionskrankh.*, 17: 167–184, 1894.

Emmerson, H.: Sunlight and Health. *Am. J. Pub. Health*, 23: 437–440, 1933.

Enders, J. F., and H. E. Pearson: Resistance of chicks to infection with influenza A virus. *Proc. Soc. Exptl. Biol. and Med.* 48: 143, 1941.

Eskey, C. R.: Fleas as vectors of plague. *Am. J. Pub. Health* 28: 1305–1310, 1938.

Eskey, C. R., and V. H. Haas: Infection in rodents, experimental transmission by fleas, and inoculation tests for infection. *Pub. Health Bull.*, Wash., No. 32, 1467, 1926.

Evans, C. A., W. M. Smith, E. A. Johnston, and E. R. Giblett: Bacterial flora of the normal human skin. *J. Investigative Dermatol.*, 15: 305–324, 1950.

Eyre, J. W. H.: The Milroy lectures on Melitensis septicemia. *Lancet* 1: 1677–1682, 1747–1752, 1908.

Falk, A.: Water-borne tularemia (incurred during handling of fish). *Minnesota Med.*, 30: 849–850, 1947.

Falk, L. L.: Bacterial contamination of tomatoes grown in polluted soil. *Am. J. Pub. Health*, 39: 1338–1342, 1949.

Farley, D. L.: Viability of ringworm fungi in dry cutaneous material, *Arch. Dermat. and Syph.*, 3: 759, 1921.

Faust, E. C.: Some facts regarding the relation between nightsoil disposal in China and the propagation of helminthic diseases. *Am. J. Trop. Med.*, 4: 487–503, 1924.

Felsen, J., and A. G. Osofsky: Gastric barrier in bacillary dysentery. *Arch. Int. Med.*, 63: 64, 1939.

Felsenfeld, O., V. M. Young, and P. Conner: Progress in the knowledge of gram negative enteric pathogenic organisms during the years from 1938–1945. *Pontifica Academia Scientiarum* 16: 1–75, 1947.

Flexner, S., and H. L. Amoss: Diffusion and survival of the poliomyelitis virus. *J. Exp. Med.*, 21: 509–514, 1915.

Flexner, S., and H. L. Amoss: Survival of the poliomyelitis virus for six years in glycerol. *J. Exp. Med.*, 25: 539–544, 1917.

Flugge, C.: Die Bedeutung der Verstreuung von Hustentropfchen fur die Verbreitung der Phthise. *Z. f. Tuberk.*, 34: 212–228, 1921.

Foa, P., and G. Gordoni-Uffreduzzi: Ueber die Aetiologie der "Meningitis cerebro- spinalis epidemica." *Z. f. Hyg.*, 4: 67–93, 1888.

Foote, M.: *News*, 66: 320, 1895.

Foshay, L., and O. B. Mayer: Viability of Bacterium tularense (Pasteurella tularensis) in human tissues. *J. Am. Med. Assoc.*, 106: 2141–2143, 1936.

Fox, J. P.: The long persistence of Rickettsia orientalis in the blood and tissues of infected animals. *J. Immunol., Virus—Res. and Exptl. Chemother.*, 59: 109–114, 1948.

Francis, E.: Laboratory studies on tetanus. *Hyg. Lab. Bull.*, U.S. Pub. Health Serv. 95: 1914.

Francis, E.: Tularemia. *Atlantic Med. J.*, 30: 337–344, 1927.

Francis, E., and G. C. Lake: Experimental transmission of tularemia in rabbits by the rabbit louse, *Haemodipsus ventricosus* (Denny). *U.S. Pub. Health Ser. Bull.*, 1–87, No. 130. 1922.

Francis, E.: Duration of viability and virulence of *Bacillusp estis. Pub. Health Repts.*, 47: 1287–1294, 1932.

Francis, T., Jr., G. Brown, and L. Penner: Search for extrahuman sources of poliomyelitis virus. *J. Am. Med. Assoc.*, 136: 1088–1092, 1948.

Frobisher, M., Jr., E. I. Parsons, S. E. Pal, and S. Hakins: A simplified method for the preservation of bacteria by desiccation in vacuo. *J. Lab. Clin. Med.*, 32: 1008–1015, 1947.

Galli-Valerio, B.: Notes de Parasitologie et de technique parasitologique. *Centr. Bact.*, 39: 230–247, 1905.

Gallo, P., T. Calderon: El virus de la neumoenteritis de los terneros. Propiddades fisicas, patogenis y cultivo. *Bol. Inst. Invest. Vet.* 3: 528–537, 1948.

Galtier, W.: Dangers des matieres tuberculeuses qui ont subi la dessication le contact prolonge de l'eau. *Compt. rend. acad. sci.*, 105: 231, 1887.

Galvagno, O., and A. Calderini: Lebensdauer und Virulenz des Typhusbacillus in Gruben, Tonnen, und im Boden. *Z. f. Hyg.*, 61: 185–208, 1908.

Gartner, A.: Uber das Absterben von Krankheitserregern im Mist und Kompost. *Z. f. Hyg.*, 28: 1–19, 1898.

Germano, E.: Die Uebertragung von Infektionskrankheiten durch die Luft. II. Mittheilung-Die Uebertragung der Diphtherie Durch die Luft. *Z. f. Hyg. u. Infektionskrankh.*, 25: 439–452. 1897.

Germano, E.: Die Uebertragung von Infektionskrankheiten durch die Luft. III. Mittheilung-Die Ueber tragung des erysipels, der pneumonie und anderga streptokokken Infektionen durch die Luft. *Z. f. Hyg. u. Infektionskrankh.*, 26: 66–89, 1897.

Gernz-Rieux, C., A. Sevin, and G. Verschoote: The survival in the skin and the spreading through the body of BCG administered by scarification in the guinea pig. *Ann. Inst. Pasteur*, 3: 103–114, 1950.

Gilbert, A., and L. Fournier: La culture du pneumocoque dans le sang defibrine. *Compt. rend. soc. biol.*, 48: 2, 1896.

Gins, H. A.: Maul- und Klavenseuche. *Klin. Wochschr.*, 3: 1135–1138, 1924.

Gladin, G. P.: Die Lesenfahigkeit der Pestbacillen unterverschieden physikalischen Bedingungen und der Einwirkung von Desenficientien. *Centr. Bakt.*, 24: 588–591, 1898.

Gould, S., L. Kaasa: Low temperature treatment of pork. Effect of certain low temperatures on viability of trichina larvae., *Am. J. Hyg.*, 49: 17–24, 1949.

Gowen, G. H.: Spirocheta in feces. *Illinois Med. J.*, 88: 202–203, 1945.

Gray, J. D. A.: The significance of *Bacterium aerogenes* in water. *J. Hyg.* 32: 132, 1932.

Green, M.: Bacteriology of shrimp. I. Introduction and development of experimental procedures. *Food Research*, 14: 365–371, 1949.

Greig, E. D. W.: On the Viability of the *Cholera vibrio* outside the human body. *Indian J. Med. Research*, Calcutta, 1: 481–504, 1913–1914.

Grubb, I., G. Laurell, K. H. Lindquist: Spread of typhoid-paratyphoid infections by means of contaminated vegetables. *Nor. Hyg. Tidskr.*, 28: 243–257, 1947.

Guarnieri, N. I.: Atti della R. Accad. med. di Roma, 4: 97, 1888.

Gunderson, T.: Germicidal effect of ultraviolet rays on virus of herpes; experimental studies. *Arch. Ophthalmol.*, 8: 519–529, 1932.

Gurevitch, J., and J. Delightisch: Survival time of *Entamoeba histolytica* in feces. *J. Palestine Jewish Med. Assoc.*, 32: 59–60, 1947.

Haagen, E.: Weitere Erfahrungen uber die Zuchtung und Konservierung des Vaksinevirus. *Zbl. Bakt.* I. 143: 283–291, 1939.

Haddow, J. R. and J. A. Idnani: The vaccination of highly susceptible animals against rinderpest. *Indian J. Vet. Sci. and Animal Husbandry* 17: 1–10, 1947.

Hall, I. W., and A. D. Fraser: The pH range for staphylococcus. *Brit. J. Exp. Path.*, 2: 242, 1921.

Hamburger, M., Jr.: Studies on the transmission of hemolytic streptococcus infections. II. Beta hemolytic streptococci in the saliva of persons with positive throat cultures. *J. Infectious Diseases*, 75: 71–78, 1944.

Hamburger, M., Jr., and M. J. Green: The problems of the dangerous carrier of hemolytic streptococci. IV. Observations upon the role of the hands, of blowing the nose, of sneezing, and of coughing in the dispersal of these microorganisms. *J. Infectious Diseases*, 79: 33–34, 1946.

Hamburger, J., Jr., L. M. Mattman, D. S. Grosch, and V. Hurst: Susceptibility to sulfadiazine of hemolytic streptococci recovered in army camps. *Am. Jr. Med.*, 1: 23–27, 1946.

Hampil, B.: The influence of temperature on the life processes and death of bacteria. *Quart. Rev. Biol.*, 7: 172–196, 1932.

Hampp, E. G.: Preservation of viability and pathogenicity of the Nichols rabbit strain of *Treponema pallidium* by freeze drying. *Publ. Health Rep.*, 66: (16) 501–506, 1951.

Hardy, A. V., M. G. Hudson, and C. F. Jordan: The skin as a portal of entry in Brucella melitensis infections. *J. Infectious Diseases*, 45: 271, 1929.

Hauptmann, W., and A. Philadelphy: Zur Schadigung des Gonokokkus durch Kalte. *Zentr. Bakt.*, 115: 186–195, 1930.

Hawley, J. E.: *The multiplication of some enteric bacteria in Musca Domestica.* Masters Thesis, Univ. of Conn. 1948.

Hellat, A.: Studies on the self-disinfecting power of the skin. *Ann. Med. Exptl. et Ciol. Fenn.*, 26: 1–95, 1948.

Henderson. W. M., and J. B. Brooksby: Survival of foot and mouth disease virus in meat and offal. *J. Hyg.*, 46: 394–402, 1948.

Heppleston, A. G.: Quantitative airborne tuberculosis in the rabbit: The course of human type infection. *J. Exptl. Med.*, 89: 597–609, 1949.

Herzberg, K.: The destruction of vaccine virus by light, energy and dyes. *Z. Immunitats.*, 80: 507–520, 1933.

Hettche, H. O.: Zur Atiologie der Rotlaufinfektion. *Arch. Hyg.*, 119: 178–183, 1937.

Hewlett, R. T.: Disinfection and disinfectants. *Lancet*, 1: 741, 815, 889, 1909.

Heymans, J. F.: La survie du bacille de la tuberculose humaine et du BCG dans le corps des bovides. *Arch. Internat. Pharmacodyn. et Therap.*, 33: 225–235, 1927.

Hill, J. H., and E. C. White: Action of normal skin on bacteria. *Arch. Surg.* 26: 901–908, 1933.

Hoffmann, E.: On syphilitic infection with material from dead bodies and possible parasitic life of Spirochaeta pallida. *Munch. Med. Wochschr.* 73: 185–187, 1926.

Homes, D., and C. S. McClesley: Bacteriology of shrimp. V. Effect of peeling, glazing, and storage temperature on bacteria in frozen shrimp. *Food Res.*, 14: 401–404, 1949.

Horder, T. J.: Bacteriology, pathology and research. *Practitioner*, 90: 15, 1913.

Horrocks, W. H.: Report of Commission on Mediterranean Fever. (On the duration of life of *M. melitensis* in unsterilized soil.) Harrison and Sons, London. 1906.

Horrocks, W. H.: On the viability and possible variation of the *Bacillus typhosus. J. Roy. Army Med. Corps*, London, 16: 225–250, 1911.

Horstmann, D. M., R. Ward, and J. L. Melnick: Persistence of virus excretion in the stools of polio patients. *J. Am. Med. Assoc.*, 126: 1061–1062, 1944.

Howe, A. A., H. A. Wen, D. Bodian, and K. F. Maxcy: Poliomyelitis virus in the human oro-pharynx. *Proc. Soc. Exp. Biol. Med.*, 56: 171, 1944.

Huddleson, I. F., H. Johnson, and E. Hamann: A study of Brucella infection in swine and employes of packing houses. *J. Am. Vet. Assn.*, 36: 16–30, 1933.

Hull, T. G.: *Diseases transmitted from animals to man.* 3rd. Ed., Charles C. Thomas, Springfield, Ill. 1947.

Hussemann, D., and M. Wallace: Studies on the possibility of the transmission of Salmonella by cooked fowl. *Food Res.*, 16: 89, 1951.

Jacotot, H.: Vitality of Pasteurella of hemorrhagic septicemia of animals. *Compt. Rend. Soc. de Biol.*, 95: 1020–1021, 1926.

Jellison, W. L., G. M. Kohls, W. J. Butler, and J. A. Weaver: Epizootic tularemia in the beaver, Castor Canadensis, and the contamination of stream water with *Pasteurella tularensis. Am. J. Hyg.*, 36: 168–182, 1942.

Jettmar, H. M. V.: Studien uber die Vitalitat der Scharlachstreptokokken. *Zeitschr. Hyg. u. Infekt.*, 107: 265–287, 1927.

Jirovec, O., and R. Peter: Uber die Resistenz der trihomonaden gegen einige Umweltfaktoren. *Schweiz. Z. Path. u. Bakt.*, 11: 146–166, 1948.

Joe, L. K.: Een eenvoudige, goedkope en doeltreffende methode voor verzending van dysenterie, typhus en paratypusfaeces naar het laboratorium. *Nederland, Tijdschr. Geneesk.*, 94: 1246–1254, 1950.

Jordan, E. O.: The changes in the bacterial content of stored normal and typhoid feces. *J. Infectious Diseases*, 38: 306–322, 1926.

Jungeblut, C. W.: Epidemic meningitis agent of disease and host resistance. Book-Fred. P. Gay. 554 p. 1935.

Kalabuchov, N., and L. Levinson: Effect of low temperature upon trypanosomes (Tryp. equiperdum) in mammals. *Nature*, 136, 553. 1935.

Kenwood, H., and E. L. Dove: The risks from tuberculous infection retained in books. *Lancet*, 2: 66, 1915.

Kessel, J. F., and C. F. Thompson: Survival of Trichomonas vaginalis in discharge. *Proc. Soc. Exp. Biol. & Med.*, 74: 755–758, 1950.

Keyes, E. L.: Observations upon the persistence of gonococci in the male urethra. *Am. J. Med. Sci.*, 143: 107–117, 1911.

King, A. G.: Viability of the organism of Rocky Mountain Spotted fever when frozen. *Jour. Infect. Dist.* 46: 279–284, 1930.

Kirby, H. J.: Observations on a trichomonad from the intestine of man. *J. Parasitol*, 29: 422, 1943.

Kirstein, F.: Ueber die Dauer der Lebenfahrigkeit von Tuberkelbacillen an flugfahrigen Staubchen. *Z. f. Hyg. u. Infektionskr.*, 50: 186–214, 1905.

Koch, R.: Die Aetiologie der Tuberculose. *Berlin Klin. Wochschr.*, 229, 1882.

Kolmer, J. A.: A note on the survival of Treponema pallidum in preserved citrated human blood and plasma. *Am. J. Syphilis, Gonorrhea, Venereal Diseases*, 26: 156, 1942.

Koser, S. A., and J. R. McClelland: Fate of bacterial spores in animal body. *Jour. Med. Res.*, 37: 259, 1917.

Kramer, S. D.: Virus of poliomyelitis; survival for over six months in feces of a contact. *J. Michigan Med. Soc.*, 39: 766–768, 1940.

Kramer, S. D., B. Hoskwith, and L. H. Grossman: Detection of the virus of poliomyelitis in the nose and throat and gastro-intestinal tract of human beings and monkeys. *J. Exp. Med.*, 69: 49, 1939.

Krantz, W.: Eine einfache Methode zur Konservierung von Rekurrens Spirochaten. *Munchener medizinische Wochschr.*, 72: 12, 1925.

Kratzeisen, E.: Ueber postmortale Spirochatenvermehrung in der Leiche. *Centr. Bakt.; abt. 1; Orig.*, 90: 91–94, 1923.

Krueger, A. P., J. W. Hope, L. R. Rosenberg, and N. S. West: Inactivation of influenza virus by human skin. *U.S. Naval Med. Bull.*, 40: 839–846, 1942.

Krumbiegel, E. R.: Transmission of virus by water. *Bull. Hyg.*, 19: 513, 1944.

Kuemen, W., and N. Swellengrebel: Die Entamoben des Menschen und ihre praktische Bedeutung. *Centr. Bakt.*, 71: 370–410, 1913.

Kusama, H.: Viability of Bacillus Dysenteriae. *Philippine J. Sci.*, 27: 1–7, 1925.

Lacy, G. R., and S. R. Haythorn: Viability of Spirocheta pallida in excised tissue and autopsy material. *Am. J. Syphilis*, 5: 401, 1921.

Landahl, H. D.: On the removal of air-borne droplets by the human respiratory tract. II. The nasal passages. *Bull. Math. Biophys.*, 12: 161–169, 1950.

Larson, W. P., and I. A. Montank: The effect of wetting on the pathogenicity and viability of the tubercle bacillus. *Proc. Soc. Exp. Biol. Med.*, 20: 229–232, 1922–1923.

Lehmann, K. B.: Das Absterben von Eiterkokken auf Linoleum Holz, Glass, und Gummi unter Beruck sichtigung von Licht und Temperature. *Arch. Hyg.*, 106: 1–31, 1931.

Leichtenstern, O.: Einiges uber Ankylostoma doudenale. *Deutsche med. Wochschr.*, 1887.

Lemon, H. M., H. Wise, and M. Hamburger, Jr.,: Bacterial content of air in army barracks. Results of a study with especial reference to the dispersion of bacteria by the air circulation system. *War Med.*, 6: 92–101, 1944.

Lepine, P., P. Sedallian, and U. Sautter: Sur la presence due virus poliomyelitique dans les mateires fecales et sa longue duree d'elimination chez un portuer sain. *Bull. Acad. de Med.*, Paris, 122: 141–149, 1939.

Levaditi, L., and A. Vaisman: Duree de conservation de la virulence de treponemes syphilomateux. *Bull. Acad. de Med.*, Paris, 130–224, 1946.

Levy, E., and H. Kayser: Uber Lebensdauer von Typhusbacillen, die im Stuhle entleert wurden. *Centr. Bakt.*, 33: 489–495, 1903.

Lidwell, O. M.: Water content of soliva dried at various humidities. *Nature*, Lond, 164: 1012, 1949.

Liston, W. G.: The plague. *Brit. Med. J.*, 1: 900–903, 950–954, 997–1001, 1924.

Lofgren, R., and M. H. Soule: The effect of low temperature on the spirochetes of relapsing fever. I. The viability of four strains of spirochetes stored at 48 C. *J. Bact.*, 50: 305–311, 1945.

Löhr, N. I.: Ueber die Bedeutung des Milieus fur das Bakterienwachstum zugleich ein Beitrag uber eine Immunisierung gegen Streptokokken und Staphylokokken. *Klin. Wochschr.*, 6: 187–188, 1927.

Lominski, I., and G. R. Thompson: Relative sterility of hands of certain metal workers. *Brit. J. Indust. Med.*, 1: 165–167, 1944.

Lösner, W.: *Arb. Reichsgesundh. Amt.*, 12: 448, 1896.

Lovell, R., M. Levi, J. Francis: Studies on the survival of *Johne's bacilli. J. Comp. Path. Therap.*, 54: 120–129, 1944.

Lowbury, E. J.: *Month, Bull. Min. Health and Emerg. Pub. Health Lab. Serv.* 2: 75, 1943.

Lu, T. H.: Cold weather resistance of contagious bacilli of digestive organs. *J. Oriental Med.*, 18: 865–874, 1933.

Luckiesch, M., A. H. Taylor and T. Knowles: Killing air-borne respiratory microorganisms with germicidal energy. *J. Franklin Inst.*, 244: 267–290, 1947.

Lu-Ti-Huan: Resistance of contagious bacilli of digestive organs against cold weather. *J. Oriental Med.*, 13: 40, 1930.

Macchiavello, A.: Survival and virulence of frozen and dried Mexican and European Typhus viruses. *Rev. Chilena de Hig. y. Med., Prev.*, 63–68. 1937.

Maccolini, R.: Multiplication potency of various species of coli-aerogenes group in water. *Boll. Soc. Ital. Biol. Sper.*, 22: 919–920. 1946.

McCormack, G.: "Pink yeast" isolated from oysters grows at temperatures below freezing. *Commercial Fish Rev.*, 12: 28. 1950.

McCullough, N. B., W. Eisele, and N. I. Pavelchek: Isolation of *Brucella abortus* from hogs. *Pub. Health Rep.*, 64: 537–538, 1949.

MacFie, J. W. S.: Observations on the role of cockroaches in disease. *Ann. Trop. Med.*, 16: 441–448, 1922.

MacGreggor, A.: The vitality of the diphtheria bacillus. *Lancet*, 1: 716, 1898.

MacKerras, I. M., and P. Pope: Experimental Salmonella infections in Australian cockroaches. *Australian J. Exp. Biol. Med. Sci.*, 26: 405–470, 1948.

McKinley, E. B., R. Fisher, and M. Holden: Action of ultra violet light upon bacteriophage and filterable viruses. *Proc. Soc. Expt'l. Bio. and Med.*, 23: 409–412, 1926.

McLeod, C.: Preservation and Inoculation studies on *Treponema pallidium*. *J. Ven. Dis. Inform.*, 30: (4) 104–107, 1949.

McNaught, J. G.: On resistance of *E. coli* to sunlight and drying. *Cent. f. bact.*, 53: 326–334, 1910.

Maddock, E. C. G.: Studies on the survival time of the bovine tubercule bacillus in soil, soil and dung, in dung and on grass with experiments on preliminary treatment of infected organic matter and the culture of the organism. *J. Hyg.*, 33: 103–117, 1933.

Malassez, N. I., and N. I. Vignal: *Compt. rend. soc. biol.*, 366, 1883.

Manson-Bahr, N. I.:

Marinelli, G., and A. Sementini: Survival of bacteria on human skin. *Rassegna internaz. Clin. e Terap.*, 27: 263–266, 1947.

Maxcy, K. F., and H. A. Howe: The significance of the finding of the virus of infantile paralysis in sewage. *Sewage Works J.*, 15: 1101–1111, 1943.

Mayer, E., and M. Dworski: Studies with ultraviolet light. III. The bactericidal action of mercury-quartz-vapor light irradiation. *Am. Rev. Tuberculosis*, 10: 166–169, 1924.

Melnick, J. L., D. M. Horstmann, and R. Ward: The isolation of poliomyelitis virus from human extra-neural sources. II. Comparison of virus content of blood, oropharyngeal washings, and stools of contacts. *J. Clin. Invest.*, 25: 275–277, 1946.

Migneco, F.: Einflub des Sonnenlichtes auf Tuberkelbazillen. *Centr. Bakt.*, 18: 729, 1895.

Migneco, F.: *Arch. Hyg.*, 25: 361, 1896.

Miles, A. A., *et al.*: Hospital infection of war wounds. *Brit. Med. Jour.* 11: 855–895, 1940.

Minett, F. C.: Sporulation and viability of B. Anthracis in relation to environmental temperature and humidity. *J. Comp. Path. and Therap.*, 60: 161–176, 1950.

Mitchell, H. J.: Further studies on ringworm of hands and feet. *Arch. Dermatol. and Syphilol.*, 5: 174, 1922.

Miyao, I.: Viability of *Spirochaeta Pallida. Phill Jour. Scin.*, 42: 199–201.

Moiser, B.: Modes of transmission of Hansen's disease. *Leprosy Rev.*, 16: 63–66, 1945.

Morse, E. V., T. Kowalczyk, and B. A. Beach: The bacteriologic aspects of experimental brucellosis in dogs following oral exposure. I. Effects of feeding aborted fetuses and placentas to adult dogs. *Am. J. Vet. Res.*, 12: 219–223, 1951.

Murayama, I.: Resistance of Spirochaeta recurrentis to temperature, light and darkness. *Okayama-Igakki-Zasshi*, 41: 152, 1929.

Nasta, M., Jovin, I., and M. Blechmann: Resistance of filterable forms of tubercle bacilli to action of different factors. *Compt. rend. soc. biol.*, 104: 723–724, 1930.

Neisser: Patholigie und Therapie der Syphillus, Berlin. *Arb. A.D.K. Gsudktsamte*, 37: 1911.

Nicholls, L., and S. A. Gunawardava: The destruction of helminth ova in night soil by composting. *Trop. Diseases Bull.*, 36: 590, 1939.

Nicola, C.: Sanchez Toledo et Veillon- De la presence du bacille du tetanos, dans les excrements du cheval et du boeuf a l'etat sain. *Centr. Bakt.*, 9: 18, 1891.

Nicoll, W.: Observations on the influence of salt and other agents in modifying the larval development of the hookworms; *Ankylostoma duodenale* and *Necator americanus. Parasitol.*, Cambridge, 9: 157–189, 1917.

Noble, W.: Experimental study of the distribution and habitat of the tetanus bacillus. *J. Infectious Diseases*, 16: 132, 1915.

Noguchi, H.: Survival of *Leptospira icterohaemorrhagiae* in nature; microchemical reactions and intermediary hosts. *J. Exp. Med.*, 27: 609, 1918.

Nohr, C. O.: Entomological background of the distribution of murine typhus and murine plague in the United States. *Am. J. Trp. Med.*, 31: 355–372, 1951.

Nolf, L. O.: Experimental studies on certain factors influencing the development and viability of the ova of the human trichuris as compared with those of the human ascaris. *Am. J. Hyg.*, 16: 288–322, 1932.

Norton, J. F., and M. F. Novy: Studies on the self-disinfecting power of the skin. *Am. J. Pub. Health*, 21: 1117, 1931.

Novikova, E. I., and G. A. Lalazarow: Viability of plague bacilli in corpses of *Citellus pygmaeus. Vestnik Mikrobiologii Epidemiologii -i- Parazitologii*, 13: 53–54, 1934.

Nussel, K.: Ueber Tuberkelbazillen befund in Stuhl. *Munchen. Med. Wochnschr.*, 70, 357–358, 1923.

Oag, R. K.: The preservation of *Borellia duttoni* by freezing. *J. Path. Bact.*, 49: 587–590, 1939.

Olesiuk, O. M.: Influence of environmental factors on viability of Newcastle disease virus. *Am. J. Vet. Res.*, 12: 43, 152–155, 1951.

Olitsky, Peter K., Casals, Jordi, Walker, Duard L., Ginsberg, S. Harold, and Frank L. Horsfall: Preservation of viruses in a mechanical refrigerator at 25° C. *Jour. Lab. and Clin. Med.*, 34: 1023–1026, 1949.

Olson, T. A., and M. E. Rueger: Experimental transmission of *Salmonella* oranienburg through cockroaches. *Pub. Health Repts.*, 65: 531–540, 1950.

Ostertag, R.: *Z. Infektkr. Haustiere*, 4: 1, 1908.

Ostrolenk, M., N. Kramer and R. C. Cleverdon: Comparative studies of enterococci and *Escherichia coli* as indices of pollution. *J. Bact.* 53: 197, 1908.

Ostrolenk, M., and H. Welch: The house fly as a vector of food poisoning organisms in food producing establishments. *Am. J. Pub. Health*, 32: 487–494, 1942.

Parr, L. W.: Sanitary significance of the succession of coli-aerogenes organisms in fresh and in stored feces. *Am. J. Pub. Health*, 26: 39–45, 1937.

Parr, L. W.: Organisms involved in the pollution of water from long stored feces. *Am. J. Pub. Health*, 28: 445–450, 1938.

Parker, E., and W. J. MacNeal: Persistence of influenza virus on the human hand. *J. Lab. Clin. Med.*, 29: 121–126, 1944.

Paul, J. R.: *Epidemiology of poliomyelitis.* A symposium delivered at Vanderbilt University, National Foundation for Infantile Paralysis, New York, 140: 1941.

Paul, J. R.: Poliomyelitis in Japan. *Am. J. Hyg.*, 45: 206–218, 1947.

Paul, J. R., and J. D. Trask: *New Internat. Clin.*, 3: 59, 1947.

Paul, J. R., and J. D. Trask: Poliomyelitis virus in urban sewage. *J. Exp. Med.*, 71: 765, 1940.

Perry, W. L.: The cultivation of *Treponema pallidium* in tissue culture. *J. Path. and Bact.*, 60: (2), 339–342, 1948.

Petri: Quoted by Ravenel in article by Webb. 1921.

Philip, C.: Observations on experimental Q fever. *J. Parasitol.*, 34: 457–464, 1948.

Price, P.: The bacteriology of normal skin. *J. Infectious Diseases* 63: 301–318, 1938.

Puntoni, V.: Survival of non sporogenous bacteria. *Policlinico (Sez. Prat.)*, 31: 219–221, 1924.

Ransom, A., and S. Delepine: *Proc. Roy. Soc.*, No. 336.

Ravitch, M. M. and J. W. Chambers: *Spirochetal* survival in frozen plasma. *Bull. Johns Hopkins Hosp.*, 71: 299–303, 1942.

Ravitch, M. M., T. W., Farmer, and B. Davis: Use of blood donors with serologic tests for syphilis—with a note on the disappearance of passively transferred reagin. *J. Clin. Invest.*, 28: 18–23, 1949.

Rebell, G., D. M. Pillsbury, M. de Saint Phalle, and D. Ginsburg: Factors affecting the rapid disappearance of bacteria placed on the normal skin. *J. Investigative Dermatol.*, 14: 247–264, 1950.

Remlinger, P. and J. Bailly: Action of freezing on Rabies virus. *Compt. Rend. Soc. de Biol.*, 116: 407–409, 1934.

Research Commission Ministry Agriculture. *Second Prog. Rep. on Foot and Mouth Dis.* 1927.

Rickard, E. R.: The survival time of the rickettsias of murine typhus in infected flea feces. *Am. J. Trop. Med.*, 31: 306–310, 1951.

Robertson, O. H.: The dispersal of respiratory pathogens in relation to the occurrence and control of airborne infections. *Am. Rev. Tuberc.*, 55: 109–123, 1947.

Robinson, Harry M., Jr.: In vitro studies of *Microsporon audouini* infection of the hair. *Arch. Derm. and Syph.*, 57: 991–997, 1948.

Rosebury, T.: *J. Immunol.*, 56: 7–96, 1947.

Rosenthal, S. R., and F. H. Elmore: Studies on the contagiousness of coccidioidomycosis. II. The fate of sphelules in sputum exposed out of doors. *Am. Rev. Tuberc.*, 61: 95–105, 1950.

Rosahn, P. D.: Infectivity of T. pallidum in excised syphilitic tissue. *Am. J. Hyg.*, 22: 283–291, 1935.

Rountree, P. M.: Gross infection of wounds in a surgical ward during a trial of the use of oiled blankets. *Med. J. Australia*, 1: 427–430, 1947.

Rubino, M. C., and Pedro Seoane: Infectiousness of meat from animals with foot and mouth disease. *Arch. Soc. Biol., Montevideo*, 1: 14–22, 1929.

Rudolfs, W., L. Falk, and R. Ragotzkie: Contamination of vegetables grown in polluted soil. II. Field and laboratory studies on Endamoeba cysts. III. Field studies on Ascaris eggs. *Sewage and Indust. Wastes*, 23: 478–485, 1951.

Sabin, A. B., and R. Ward: Natural history of human poliomyelitis. II. Elimination of the virus. *J. Exp. Med.*, 74: 519–529, 1941.

Sanfelice, F.: Untersuchungen uber anaerobe Mikroorganismen. *Z. Hyg. Infektionskrankh.*, 14: 339–392, 1893.

Saunders, G. M., and V. Scott: Preservation of Plasmodium vivax by freezing. *Science*, 106: 300–301, 1947.

Savage, W. G., and D. R. Wood: The vitality and viability of streptococci in water. *J. Hyg.*, 16: 227–239, 1917.

Sawers, W. C.: Some aspects of the Leptospirous problem in Australia. *Med. J. Australia*, 25: 1089–1097, 1938.

Sawizky, N. I.: *Zur Frage uber die Dauer der infektiosen Eigenschaften des getrockneten tuberculosen Sputums. Dissert, Inaug. St. Petersburg.* 1891.

Sawyer, W. A., W. D. M. Lloyd, and S. F. Kitchen: The preservation of yellow fever virus. *J. Exp. Med.*, 50: 1, 1929.

Schalk, A. F.: Results of some avian tuberculosis studies. *J. Am. Vet. Med. Assoc.*, 72: 852–864, 1928.

Schiemann, O., H. Landau: Uber Handedesinfektion und Handereinigung in ihrer Bedentung zur Verhutung von Krankeitsubertragungen. *Z. Hyg. Infektionskrankh.*, 88: 129–184, 1919.

Schill, N. I., and N. I. Fischer: Ueber die Desinfection des Auswerfes der Phthisiker. *M. Kais. G. A., Bd.* 11, 133, 1884.

Schiller, N. I.: Zum Verhalten der Erreger der Cholera und des Unterleibs typhus in dem Inhalt der Abtrittsgruben und Abwasser. *Arbeiten aus D. K. Gesundhtsamte*, 6: 197–208, 1890.

Schofield, F. S.: Effects of temperature on gonococci. *J. Urol.*, 17: 581–591, 1927.

Schottelius, N. I.: Ueber das Verhalten der Tuberkilbacillen im Erdboden. *Deutsche Med. Wochschr.*, 16: 226, 1890.

Selbie, F. R.: Viability of Spirochete Pallida in stored plasma. *Brit. Jour. Exptl. Path.*, 24: 150–152, 1943.

Sellards, A. W., and E. Hindle: The preservation of yellow fever virus. *Brit. Med. J.*, 1: 713, 1928.

Semple, D.: The relation of tetanus to the hypodermic or intramuscular infection of Quinine. *India Govt. Sci. Mem.* 43: 1911.

Sergent, A.: Fievve Recurrente a Spirocheta Hispanicum en Algerie transmission par le Rhipicephale du chien premunition serumde convelescents. *Ann. Inst. Pasteur*, 61: 217–254, 1938.

Shpuntoff, H., and T. Rosebury: Infectivity of fusospirochetal exudates for guinea pigs, hamsters, mice and chick embryos by several routes of inoculation. *J. Dental Res.*, 28: 7–16, 1949.

Shu-Hsian, C.: Die lebensdauer von Rockettsia in Lausefaeces. *Schweiz. Z. Path. u. Bact.*, 12: 508–512, 1949.

Sichert-Modrow, I.: Vitality of causative agent of Foot and Mouth disease at various temperatures. *Cen. f. Bakt.*, 119: 17–19, 1930.

Singer, C., and Arnold, L.: Auto-disinfecting power of the human skin. *Proc. Soc. Exp. Biol. Med.*, 27: 364, 1929.

Skidmore, L. V.: The transmission of fowl cholera to turkeys by the common house fly, with brief notes on the viability of fowl cholera microorganisms. *Cornell Vet.*, 22: 281–285, 1932.

Smith, C. R.: Survival of tubercle bacilli. The viability of dried tubercle bacilli in unfiltered room-light, in the dark and in the refrigerator. *Am. Rev. Tuberc.*, 45: 334–345, 1942.

Smith, C. R.: Survival of tubercle bacilli in books; how contaminated boils may be rendered uninfectious. *Am. Rev. Tuberc.*, 46: 549–559 1942.

Smithburn, K. C., A. F. Mahaffy, A. J. Haddow, S. Kitchen, and J. F. Smith: Rift Valley fever. Accidental infections among laboratory workers. *J. Immunol., Virus Res. and Exp. Chemother.*, 62: 213–227, 1949.

Smyth, H. F., E. F. Pike: Disinfecting skins and hair for Anthrax. *Amer. Jour. Hyg.*, 1: 541–555, 1921.

Soparker, M. B.: Vitality of tubercle bacillus outside the body. *Indian J. Med. Res.*, 4: 627, 1917.

Sormani, G.: Contribuzione agli sulla storia naturale del bacillo tuberculare. *Estr. dal Boll. Scient.* No. 1. 1886.

Sormani, G.: *Centr. Bakt.*, 9: 421, 1891.

Spector, B., and F. Buky: Viability of *Endamoeba histolytica* and *Endamoeba coli*. Pub. Health Repts., 49: 379–385, 1934.

Spink, W. W., and D. Anderson: Brucella studies on bank blood in a general hospital. A. Agglutinins; B. Survival of Brucella. *J. Lab. and Clin. Med.*, 35: 440–445, 1950.

Starzyk, J.: Vitalite, toxicite et pouvoir d'immunisation de *Rickettsa prowazeki* conservees hors de l'-organisme du pou, en milieu liquide et en milieu sec. *Compt. rend. soc. biol.*, 123: 1221–1225, 1936.

Stavitsky, A. B.: Preservation of *Leptospira icterohemorrhagiae* in vitro. *J. Bact.*, 50: 118, 1945.

Stein, C. D.: Some observations on the tenacity of *Bacillus anthracis*. *Vet. Med.*, 42: 13–22, 1947.

Stenhouse, R., R. Williams, and W. Hoy: The viability of B. tuberculosis on pasture land, in stored feces and liquid manure. *J. Hyg.*, 30: 412, 1930.

Sternberg, G. M., and J. T. Dezendorf: The action of sunlight on microorganisms. *Med. Rec., N.Y.*, 16: 607, 1894.

Stewart, W., Major: Viability of dysentery bacilli and transmission by flies in North Africa. *J. Roy. Army Med. Corps.*, 83: 42–46, 1944.

Stewart, A. D., and S. C. Ghosal: On the value of Wilson and Blair's bismuth sulphite medium in the isolation of *B. typhosus* from feces and sewage. *Ind. J. Med. Res.*, 20: 341–345, 1932–1933.

Stillman, E. G.: The viability of pneumococci in dried rabbit blood. *Jour. Infect. Dis.*, 66: 171–173, 1940.

Stockman, S., and F. C. Minett: *Jour. Comp. Path. and Therap.*, 39: 1–30, 1926.

Stone, A. K.: Why the sputa of tuberculous patients should be destroyed. *Centr. Bakt.*, Bd. 10: 106, 1891.

Strong, R. R.: *Prevention and treatment of tropical diseases.* 6th Ed., Blakiston, Philadelphia *(Stitt's Diagnosis)* 1942.

Sweany, H. C., and C. C. MacLane: Relation of dust to spread of tuberculosis. *Illinois Med. J.*, 36: 302–305, 1919.

Swift, B. H.: *Trichomonas vaginalis* vaginitis as a cause of pruritus vulvae. *Med. J. Australia*, 1: 123, 1937.

Tarozzi, G.: Ueber das latent Leben der Tetanussporen im tierischen Organismus. *Centr. Bakt., Abt.* 1, Orig., 40: 305–311, 451–458, 1906.

Teague, O., and M. A. Barber: Studies on pneumonic plague and plague immunization. *Philippine J. Sc.*, 7: 255–268, 1912.

Theiler, M.: Studies on the action of yellow fever virus in mice. *Ann. Trop. Med. Parasitol.*, 24: 249–272, 1930.

Thomas, S. and W. A. Kreidler: The effect of freezing on *Eberthella typhi. Abstracts Bact.*, 9: 25, 1925.

Tohyama, Y.: Experimental investigations concerning the prevention of epidemic diseases due to aquatic products. *Sci. Repts. Govt. Inst. Infectious Diseases*, Tokyo, 4: 169–222, 1925.

Toma, de P.: Sulla virulenza dello sputo tuberculare. *Ann. di Med.*, 275, 3., 1886.

Tonney, F. O., and R. E. Noble: Relative persistence of Bact., *coli* and aerogenes in nature; in decayed stumps. *J. Bact.*, 22: 433–446, 1931.

Toomey, J. A., Takacs, and M. Schaeffer: Attempts to infect amoeba proteus with poliomyelitis virus. *Am. J. Diseases Children*, 75: 11–14, 1948.

Topping, N. H.: The preservation of the infectious agents of some of the rickettsioses. *Pub. Health Repts.*, 55: 545–547, 1940.

Tovar, R. M.: Infection and transmision of Brucella by ectoparasites. *Am. J. Vet. Res.*, 8: 138–140, 1947.

Toyoda, H.: Relapsing fever. *Nat. Med. J. China*, 17: 233–267, 1931.

Trask, J. D., J. R. Paul, and A. J. Vignec: Poliomyelitis virus in human stools. *J. Exp. Med.*, 71: 751–763, 1940.

Trask, J. D., J. R. Paul, and A. J. Vignec: Observations on fecal examination in poliomyelitis. *Am. J. Pub. Health*, 31: 239–244, 1941.

Trask, J. D., A. J. Vignec and J. R. Paul: Poliomyelitis virus in human stools. *J. Am. Med. Assoc.*, 3: 6, 1938.

Turner, T. B., and N. L. Brayton: Factors influencing the survival of spirochetes in the frozen state. *J. Exp. Med.*, 70: 639–650, 1939.

Turner, T. B., and T. H. Diseker: Duration of infectivity of *Treponema pallidum* in citrated blood stored under conditions obtained in blood banks. *Bull. Johns Hopkins Hosp.*, 68: 269, 1941.

Turner, T. B., and W. L. Fleming: Prolonged maintenance of spirochetes and filterable viruses in the frozen state. *J. Exp. Med.*, 70: 629–637, 1939.

Twichell, D. C.: The vitality of tubercle bacilli in sputum. *Med. News., N.Y.*, 87: 642–647, 1905.

Twichell, D. C.: Vitality of tubercle bacilli in sputum. *Trans. Nat. Assn. for Study and Prev. of Tuberculosis.* 1: 221, 1906.

Uffelman, J.: Die Dauer der Lebensfahigkeit von Typhus und Cholerabacillen in Faralmassen. *Centr. Bakt.*, 5: 497–502. 1889.

Uhlenhuth, N. I., and N. I. Fromme: Zur Aetiologie der sogenannten Weil'schen Krankheit (Ansteckende Gelbsucht). *Berlin. Klin. Wochschr.*, 53: 269–273, 1916.

Ungerman: *Arb. Kaiserl. Gesundheitsamt.* 51: 199, 1918.

Urbain, A., G. Guillot, and M. Vallee: Longevity of *Preisz-Nocard Bacillus. Compt. Rend. Soc. de Biol.* 105: 676–677, 1930–1931.

Vacek, B.: Examination of some conditions necessary for the survival of the typhoid bacillus in water. *Casopis Ceskoslov. Lekarnictva.* 71: 97, 1932.

Vasquez-Oolet, A.: Viability of intestinal pathogenic bacteria in fruits and Philippine foods eaten raw. *Philippine J. Sci.* 24: 35–39, 1924.

Vidal, C. B.: Viability of tubercle bacilli in tissues and sputum preserved in vitro. *Am. Rev. Tuberc.*, 30: 345, 1934.

Villemin, N. I.: De la propagation de la phtisie. *Memoire Lu a l'Acad. de Med.* 1869.

Welch, H., M. Ostrolenk, and M. T. Bartram: Role of rats in the spread of food poisoning bacteria of the Salmonella group. *Am. J. Pub. Health,* 31: 332–340, 1941.

Webb, G. B., C. T. Ryder, and G. B. Gilbert: The survival and virulence of Tubercle Bacilli in excised animal lymph nodes. *Amer. Rev. Tuberc.* 5: 388–396, 1921.

Wells, W. F.: Seasonal variations in multiplication rate of microorganisms within the body. *Am. J. Pub. Health,* 11: 636–638, 1921.

Wenrich, D. H.: Culture experiments on intestinal flagellates. V. Some additional logevity records to September 1, 1949. *J. Parasitol.*, 35: 32, 1949.

Wenyon, C. M., and F. W. O'Connor: *Human intestinal protozoa in the Near East.* London, Wellcome Bureau of Scientific Res. 1917.

Whitwell, G., and I. Sutherland: Boils and infected hands: an epidemiological investigation. *Brit. J. Ind. Med.*, 5: 88–92, 1948.

Williams, R. S., and W. A. Hoy: Viability of *B. tuberculosis* (Bovinus) on pasture land, in stored feces, and in liquid manure. *Jour. Hyg.*, 30: 413–419, 1930.

Winter, C. E., and L. A. Sandholzer: Studies of the fecal streptococci. I. The isolation of enterococci from natural sources. *Fish. Leafl.* 201: 1–6, 1946.

Wood, F. C.: The viability of the pneumococcus after drying: a study of one of the factors in pneumonic infection. *J. Exp. Med.*, 7: 592–625, 1905.

Wynns, H. C., and M. D. Beck: Epidemiological studies on relapsing fever in California. *Am. J. Pub. Health,* 25: 270–276, 1935.

York, W., and A. Adams: Observations on *Entamoeba histolytica. Ann. Trop. Med. Parasitol.*, 20: 317–326, 1926.

Yoshida, S.: On the resistance of Ascaris eggs. *J. Parasitol.*, 6: 132–139, 1920.

Zeller, H.: Untersuchungen uber das Wachstum des Typhus-bacillus im Harn. *Z. Hyg. u. Infektionskrankh.* 128: 591–615, 1948.

Zina, G.: Ricerche sull 'Indice microbico cutaneo negli adulti. *Giorn. Bacteriol. e Immunol.*, 38: 407–420, 1948.

Zurhelle, E., and R. Strempel: Studien uber Lebensfahigkeit und Virulenzerhaltung der Spir. pall. in totem Gewebe. *Arch. Dermatol. u. Syphilol.*, 153: 219–226, 1927.

HISTORICAL ASPECTS AND PRESENT DAY STATUS OF THE RADICAL BREAST OPERATION FOR CANCER*

JOSEPH K. JOHNSON

The development of the modern operation for mammary cancer forms one of the noblest chapters in surgical history. Many of the principles which are now applied in all fields of cancer surgery have been learned in the study of this lesion. In this era of critical appraisal of the results achieved by radical surgery for breast carcinoma it is both reassuring and enlightening to look back at the progress which has been made in the treatment of this exceedingly prevalent disease.

As surgeons it is difficult for us to take great pride in the accomplishment of our tonsorially trained predecessors of centuries past. Nevertheless, the surgeons of antiquity were familiar with the manifestations and natural history of breast cancer, and their efforts at its eradication, brutal though they were, may have offered the cancer victim some advantage—either by prolonging life considerably or by terminating it abruptly.

Recognizable descriptions of the clinical manifestations of mammary cancer can be found in such ancient records as the Edwin Smith surgical papyrus, the Ebers papyrus, the code of Hammurabi, the Rig-Veda and Atharva-Veda. It is difficult to determine from these writings whether any treatment other than salves, poultices and fomentations was ever resorted to in the case of true malignant disease. The Ebers papyrus, (*circa* 1500–1600 B.C.) however, describes a "fatty tumor" of the breast successfully treated "with the knife."

Mastectomy was not practiced widely during the first millenium. Roman surgeons of this period occasionally resorted to heroic surgery for advanced breast cancer, and Celsus in the 1st century A.D., advocated simple mastectomy for early lesions. Celsus cautioned particularly against removal of the pectoral muscles, and also warned of the inefficacy of all medicinal therapy. It is recorded that Leonides, an Alexandrian surgeon

* From the Department of Surgery, The University of Texas Medical Branch, Galveston. Accepted for publication, September 9, 1955.

of some fame during the first century, rather meticulously excised the breast with the knife, following closely with the cautery to secure hemostasis. Galen, during the second century, gave an extremely accurate description of breast cancer, together with surgical methods for its treatment. He wrote: "In all operations we attempt to excise a pathologic tumor in a circle, in the region where it borders on the healthy tissue. On account of the size of the vessels. . . . there is immediate danger of hemorrhage, but if you use ligatures extension of the disease to the surrounding parts takes place."[1] Paul of Aegina, last of the Greek eclectics, gives a similar description of the surgical approach to breast cancer in the Sixth Book of his "Epitome."

During the middle ages little of merit was contributed to the treatment of this disease. Ambroise Paré (1510–1564) was essentially a Galenist, removing only small, non-ulcerated breast tumors which were readily accessible. He was among the first to recognize, however, the relationship between breast cancer and its axillary extensions. In 1562 Vesalius published a description of an operation for the removal of the cancerous breast. He employed wide excision of skin and breast tissue and employed ligatures to control bleeding. Severinus (1580–1656) of Salerno favored excision of benign breast tumors as prophylaxis against the occurrence of malignancy. He was one of the first surgeons to excise the enlarged axillary nodes at the time of mastectomy.

From what is known of this ancient surgery it is probable that such operations were infrequently performed and were done more in a spirit of desperation than in the hope of cure. Some of the best surgeons of the late eighteenth and early nineteenth centuries were able to report an occasional case which was apparently cured by surgery. Nelaton, for example, reported several such cures. Velpeau, in his "Treatise on the Diseases of the Breast," published in 1856, knew of seven women of a group of 187 who were alive from 5 to 20 years postoperatively. Velpeau advised excision of the pectoral muscles when the cancer appeared to be grossly invading the fascia. He did not routinely amputate the breast, however, preferring to do only wide local excisions.

[1] Cited by Lewison (1953).

In general, this period is characterized by an attitude of hopelessness. The spirit of the day was well expressed by Hayes Agnew in a lecture delivered shortly before his death. He stated that he operated on patients solely for the effect on their morale. It was his honest conviction that surgery shortened rather than prolonged their lives. Typical of the gloomy view on the prognosis in breast cancer were the opinions expressed by two leading authorities. Adolph Bardeleben, in a textbook in 1865, and Sir James Paget, in 1870, both stated categorically that carcinoma of the breast was not curable by surgical means. It is interesting to note that James Syme, in his "Principles of Surgery," published in 1842 was an advocate of wide excision for early cases of breast cancer. He also recommended axillary lymph node dissection as a part of the procedure.

It was not until the pathologic anatomy and mode of dissemination of mammary cancer was elucidated that any really scientific approach to the problem of treatment could be made. Rudolph Virchow, the great German pathologist, stimulated much thought in the minds of surgeons with his *Omnis cellula e cellula* concept of tumor formation. Carl Ruge, a Berlin gynecologist, introduced the idea of obtaining surgical biopsy material for positive microscopic diagnosis. Although Ruge employed this method for the diagnosis of uterine cancer in 1878 it was not until the early 1890's that the technique was adopted in this country by Howard Kelly at Johns Hopkins. Julius Cohnheim, in 1865, invented a method of preparing frozen tissue sections for microscopic study. This advance found little practical application in this country until L. B. Wilson of the Mayo Clinic popularized the method.

That progress in the field of pathology has served as a great stimulus and help in the development of the surgical treatment for cancer of the breast cannot be denied. Carl Thiersch, a German surgeon, showed, in 1865, that carcinoma of the skin arising from a single site may spread to distant foci by way of the lymphatic and blood vascular systems. Wilhelm Waldeyer, in 1872, was able to confirm this fact in relation to cancer of the breast and internal organs. The impact of this concept on surgeons of that period cannot be over-estimated when it is recalled that much of the philosophy of hopelessness resulted from the

belief that widespread cancer arose primarily in each of the organs and sites involved. Who would undertake an operation for the cure of a disease thought to be generalized from the outset?

In 1867 Charles H. Moore of the Middlesex Hospital in London advocated the *en bloc* removal of the entire breast and axillary contents. His operation contained the essence of the modern radical mastectomy except for the important inclusion of the pectoral muscles. Though his principle received scant attention in England where the views of Paget currently prevailed they were quickly picked up in Germany and in America. Richard von Volkmann, professor of surgery at Halle, made the observation from microscopic studies that the fascia of the pectoral muscles was very frequently invaded by tumor. He began the routine removal of the pectoral fascia in addition to *en bloc* resection of the breast and axilla. His views were strongly supported by Heidenhain, and this operation soon became standard in great clinics of Germany and Austria. Within a few years it was possible to compile statistics on significant groups of surgically treated cases of breast cancer. These statistics, based on microscopically proven cases of mammary carcinoma, proved for the first time that surgical cure could be effected in an appreciable number of cases.

Unfortunately the enthusiasm generated by Volkmann for his semi-radical operation for breast cancer was considerably dimmed in subsequent years when the incidence of local recurrences and distant metastases became more fully appreciated. Volkmann, at one time, had optimistically reported that a woman, alive and free of cancer 12 months after mastectomy, could be considered as "cured."

In America, Joseph Pancoast of the Jefferson Medical College was among the first in this country to advocate *en bloc* mammary and axillary node resection. He did not, however, divide the pectoral muscles. Samuel W. Gross, in his book on "Tumors of the Mammary Gland," published in 1880, advocated the operation of C. H. Moore, and, like Volkmann, he extended it to include removal of the pectoral fascia. Halsted, in New York, was at this time (1882) routinely cleaning out the axilla in all cases of breast cancer. Having closely followed the work of Volkmann

and having noted the extremely high local recurrence rates reported from the German and Austrian clinics, Halsted became convinced that the removal of the pectoral fascia alone was insufficient. He accordingly began the routine excision of the pectoralis major. The pectoralis minor, however, was merely cut across to permit wide excision of fat, connective tissue and lymphatics. It is strange that he felt that preservation of the pectoralis minor was of any importance, however, he did not begin to remove this muscle until several years later.

So great was Halsted's contribution to the surgical treatment of mammary cancer and such was his influence upon the course of American surgery that some mention must be made of his career and the era in which he lived. A graduate of the College of Physicians and Surgeons in New York, Halsted served for more than a year as interne and house physician in New York hospitals (Bellevue and New York Hospital). In 1878 he went abroad, spending two years visiting and studying in the surgical clinics of Germany and Austria. He was greatly impressed by the skill and daring of such surgeons as von Bergmann, Billroth, Mikulicz, Kolliker, Stoher, Wigert, and von Volkmann. Following his return to New York in 1880 Halsted launched upon a brilliant surgical career. In the period from 1882 to 1886 alone he published 21 scientific papers. His studies included pioneer work on the treatment of appendicitis (or perityphilitis as it was then called), traction and countertraction in the treatment of fractures, blood transfusion, and regional anesthesia. In 1881 he saved his sister's life by giving her the first recorded direct syringe blood transfusion (using his own blood). A year later he performed one of the first successful cholecystectomies on his mother.

By 1885 Halsted was enjoying great success and popularity in New York social and surgical circles. It was at this time that he and his two associates, Hall and Hartley, began their studies on regional anesthesia. Cocaine was then a relatively new drug, the anesthetic properties of which had been reported by Koller at the Ophthalmic Congress in Heidelberg. Nothing was known of its addictive powers, consequently the three surgeons, together with some medical students with whom they were working soon fell victim to the terrible effects of the drug. Dr. William H.

Welch, pathologist at Bellevue and devoted friend of Halsted, was quick to recognize the gravity of the situation. He is said to have hired a schooner and sailed with Halsted to the Windward Islands and back. On their return to Providence, R. I., Halsted entered the Butler Hospital where he stayed for several months. Records of the hospital, according to Whipple (1952), indicate that he was free from all evidence of his addiction when he left the hospital. This experience created a profound change in Halsted's personality. From extreme extrovertism he became a marked introvert, almost a recluse. Whipple feels that Halsted's addiction and subsequent cure was most fortunate for American Surgery. Certainly his change in personality made him a much more meticulous, careful surgeon and a profound scholar. In 1889 Halsted and Welch both left New York to join the staff of the New Johns Hopkins Hospital which was just opening. There he remained until his death in 1922.

In 1894 Halsted presented results of his complete operation for the cure of breast cancer. As has been mentioned, this operation did not include excision of the pectoralis minor which was merely divided to facilitate removal of fat and connective tissue containing lymphatics. In 1898, however, Halsted wrote that he was almost routinely cleaning out the supraclavicular region and by this time he routinely excised the pectoralis minor as well as the major. Having witnessed the very frequent local recurrence of breast cancer following the operations performed by his contemporaries, it was Halsted's principle to excise considerable areas of skin and to cut very thin skin flaps. In order to keep subconscious concern for the closure from preventing him from doing an adequate resection he often left the room as soon as the dissection was complete. An assistant would then close the wound, almost always using skin grafts.

It is of great importance to note also that at this time Bloodgood, who was Instructor in Surgery, had extended Halsted's dissection into the neck and in one instance into the mediastinum. Cushing, Halsted's house surgeon, had at this time cleaned out the mediastinum in three cases of recurrent disease. Halsted ventured to predict that in the future he might remove the mediastinal lymphatics in some of his primary operations. He also practiced stripping the sheaths of the rectus, serratus

anterior, subscapularis, latissimus dorsi and teres major in extensive cases.

The same year that Halsted published his second description of the radical mastectomy Willy Meyer presented his improved operation for breast cancer. Both men had independently conceived an essentially similar approach. Meyer's operation began in the axilla and involved the *en bloc* removal of the breast and both pectoral muscles. Meyer's operation was conceived independently and differed from Halsted's only in skin incision and direction of dissection. He reported only 6 cases, however, and these only over a 3 year period. Halsted, on the other hand, had over 50 cases, complete with follow-up, over a 12 year period.

The chief advances in breast surgery since the turn of the century have been in the direction of improving operability and in decreasing surgical mortality. The splendid job done by the American Cancer Society in educating the public has contributed toward increasing the number of cases which are operable when first seen. Adair (1953), for example, has reported that in 1920 only 22.6 per cent of women with breast carcinoma seen at Memorial Hospital were operable. By 1951, 80.3 per cent of women seen on the same service were considered operable. Haagensen and Stout (1943) have recognized the fact that certain cases of mammary cancer are categorically inoperable when first seen. Their criteria for determining operability cannot be followed too rigidly, however, as an occasional "inoperable" case may be cured by radical surgery.

With regard to the improvements in operative mortality it cannot be denied that survival statistics are considerably improved by the elimination of operative deaths. Recent advances in anesthesia, blood replacement and antibiotic control of infection must be credited for their share in increasing the effectiveness of surgery for the cure of cancer.

Unfortunately, there is still a considerable group of cases of breast cancer, operable by the usual standards, for which the standard radical operation cannot be curative. Handley, on the basis of random biopsies of the second and third intercostal spaces, has found 34 per cent to have positive nodes at the time of operation. Wangensteen states that approximately 60 per cent of patients with axillary metastases are beyond the reach of the

conventional radical mastectomy. He advocates a more extensive radical procedure. Urban has developed a technique for excising the anterior chest wall on the affected side. It is too soon to evaluate the results of these super-radical operations.

The life of the incurable cancer patient has been prolonged and rendered more comfortable in recent years by the improved application of radiotherapy. Hormonal control also has effected dramatic benefits in selected cases. Castration and adrenalectomy as adjunctive procedures in certain cases of acinar or lobular adenocarcinoma of the breast are being employed with greater frequency. It must be emphasized, however, that these are purely palliative procedures.

The efficacy of the radical breast operation is extremely difficult to assess with any great accuracy. Statistical methods which have been employed are not too reliable. Park and Lees (1951), for example, concluded that survival rate in breast cancer may not be influenced by any form of treatment. Gordon-Taylor (1948) on the other hand, has reported 84 per cent ten-year survival in his stage I carcinoma (43 per cent for all stages in his 388 cases). Geschickter's figures, based on a comparative review of the American literature, indicate a five-year survival rate of 36.9 per cent in a total of 8,585 cases treated surgically. By way of comparison, Nathanson and Welch (1936) reported in 1936 on 100 cases of breast cancer which received no treatment. At the end of five years 19 of these women were still alive; at the end of ten years only six patients were still living and all six were in advanced stages of the disease. In contrast are Haagensen's figures on women operated on for breast cancer at Presbyterian Hospital in the years between 1915 and 1938. At the end of five years 50 per cent were alive, and at the end of ten years 31 per cent were alive and apparently free of cancer. Such figures, when corrected by the normal life expectancy curve, offer convincing proof of the value of the radical breast operation.

McWhirter, using special radiation techniques combined with simple mastectomy, has reported 40.2 per cent five year survival in a considerable group of unselected cases. These figures have not been duplicated by other radiotherapists, though they certainly offer a challenge. There is, moreover, no convincing proof

that patients thus treated are actually cured. The ten year follow-up study on this group of patients will be much more revealing as regards the true efficacy of this method.

From this review it may be concluded that the radical breast operation, as conceived by Halsted and Meyer continues to be, as it has for the past fifty years, the accepted treatment for the cure of breast cancer. Patients with early breast cancer, as exemplified by those falling into the Manchester stage I group, have an excellent chance for cure. Those with more advanced disease, exclusive of the categorically inoperable cases, have less chance of cure but a greater life expectancy may be anticipated, especially where adjunctive therapy is utilized.

REFERENCES

Adair, F. E.: Cancer of the Breast. *S. Clin. North America,* 33: 3, 1953.

Gordon-Taylor, G.: Discussion: The treatment of Carcinoma of The Breast. *Proc. Roy. Soc. Med.*, 41: 118, 1948.

Haagensen, C. D., and A. P. Stout: Carcinoma of the Breast; Criteria of Operability. *Ann. Surg.*, 118: 859, 1943.

Lewison, E. F.: The Surgical Treatment of Breast Cancer, An Historical and Collective Review. *Surg.*, 34: 904, 1953.

Nathanson, I. T. and C. E. Welch: Life Expectancy and Incidence of Malignant Disease. I. Carcinoma of the Breast. *Am. J. Ca.*, 28: 40, 1936.

Park, W. W., and J. C. Lees: The Absolute Curability of Carcinoma of the Breast. *S. G. and O.*, 93: 129, 1951.

Whipple, A. O.: Halsted's New York Period. *Surg.*, 32: 542, 1952.

THE EFFECT OF CALCIUM ON CHROMOGENESIS IN *MICROCOCCUS PYOGENES*•

JAMES A. JOHNSTON

The carotenoid pigment of *Micrococcus pyogenes* is produced slowly and in poor yield on common semi-solid media. Fujita and Yoshioka (1923) noted that the addition of 10 per cent evaporated milk to a basal medium stimulated pigment production. Using a Difco Proteose Peptone No. 3 agar fortified with 0.5 per cent yeast extract, it was noted in this laboratory that pigmentation occurred only when milk was added. The growth on the basal medium was equal in amount to that on milk agar, but the colonies were white to gray in color. Experiments were begun to determine the factor in milk which stimulated pigment formation, and the results are reported here.

Materials and Methods

The test organism used in all experiments unless otherwise specified was *Micrococcus pyogenes* var. aureus, strain No. 139, which was coagulase, gelatinase, and mannitol positive. All of the strains of *M. pyogenes* studied were isolated from ophthalmic infections and identified by Mrs. M. L. Sigtenhorst.

The basal medium, Proteose Peptone No. 3 Agar, contained per liter 20 g. Proteose Peptone No. 3 Difco, 0.5 g. dextrose, 5 g. yeast extract Difco, 5 g. sodium chloride, 5 g. disodium phosphate, and 20 g. agar Difco. The pH was adjusted to 7.3 before autoclaving. Stock cultures were maintained on basal medium supplemented with 10 per cent (v/v) evaporated milk, "Carnation" Brand, and held at room temperature, approximately 25° C. For pigmentation experiments agar slants were inoculated with constant amounts of dense aqueus suspensions prepared from 36–48 hour agar slant cultures. All cultures were incubated approximately 18 hours at 37° C. and 18 hours at room temperature before reading. The amounts of pigment and growth were judged visually.

The purification of casein was done according to the procedures of Warner (1944).

Results and Discussion

Crude fractionations of milk indicated that the pigment factor was in the casein fraction. Commercial purified caseins and

• From the Department of Bacteriology and Parasitology, The University of Texas Medical Branch, Galveston. Accepted for publication, August 8, 1955.

casein hydrolysates, acid and enzymic, would not support pigment formation. The stimulation by freshly prepared caseins became progressively smaller as the casein was purified by isoelectric precipitations. The supernatant from the casein purifications were stimulatory to variable degrees.

The purification of casein would remove carbohydrates and metallic ions. Of the several carbohydrates tested, glucose, sucrose, and lactose, none was effective. As may be seen in table 1, calcium ions were stimulatory to pigment formation.

TABLE 1

The Effect of Calcium Salts on Pigment Formation

Ca mg./cc.	Salt		
	Lactate	Gluconate	Chloride
0	—	—	—
0.5	+	++	±
1.0	++	+++	++
1.5	++	++++	+++
2.0	++	++++	++
2.5	++	+++	±
3.0	++	+++	±
3.5	++	++	±
4.0	++	++	±

M. pyogenes No. 139 was incubated 18 hours at 37° C. and 18 hours at 25° C. Degree of pigmentation is indicated by + to ++++ or no pigment by —.

Three calcium salts, lactate, gluconate, and chloride, were studied. Regardless of the anion present, calcium stimulated pigment formation. However, the optimal concentrations of the several calcium compounds varied with the anion used. The addition of calcium to the basal medium, regardless of the salt form, caused the formation of a precipitate. This precipitate was probably the calcium salts of dicarboxylic amino acids. Hence it was clear that in the complex system studied the effective concentration of calcium was dependent upon the calcium binding of the anion used and of the amino acids in the medium. When a sequestering agent such as ethylenediamine tetracetic acid was added, no calcium effect was noted presumably due to the high affinity of that compound for calcium.

Removal of the precipitate from the calcium containing medium also removed the pigment-stimulatory effect as shown in table 2. Chromogenesis occurred upon the addition of more

calcium. The action of calcium then appeared to be direct since this experiment ruled out the possibility that calcium formed a gel which absorbed substances from the medium which inhibited pigment formation. Such an effect has been demonstrated by Altman and Crook (1953) for succinoxidase.

TABLE 2

Fraction of the Calcium Medium

Basal medium + Ca (1.5 mg./cc.)		Growth, Color
CENTRIFUGE		
Precipitate	Supernatant*	Growth, No Color
	Supernatant + Ca (1.5 mg./cc.)	Growth, Color

*Shows no freee calcium when tested with oxalate.

Other cations were tested for their effect upon chromogenesis. These included barium, magnesium, zinc, manganese, copper, ferric iron, cobalt, sodium and potassium. None of these metals was stimulatory, and magnesium had an inhibitory effect for the test strain as may be seen in table 3. When the medium

TABLE 3

The Effect of Calcium and Magnesium on Pigment Formation
Each tube contains 1.0 mg. Ca/ml.

Mg. mg./cc.	Color
0.1	++++
0.2	+++
0.4	++
0.6	+
0.8	+
1.0	+
1.2	±

M. pyogenes No. 139 was incubated 18 hours at 37° C. and 18 hours at 25° C. Degree of pigmentation is indicated by + to ++++. Calcium was supplied as $CaCl_2$.

contained a constant, optimal amount of calcium, addition of increasing quantities of magnesium caused a corresponding decrease in pigment formation. The same effect was observed when the ionic strength of the medium in all tubes was maintained constant with either sodium or potassium chloride. It should be

noted, however, that magnesium did not antagonize the calcium effect in all strains tested.

The effect of calcium on pigmentation in additional strains of three varieties of *M. pyogenes* was tested. The results are given in table 4. Forty-eight colored strains and 31 white ones were

TABLE 4

The Effect of Calcium on Several Strains of Micrococcus Pyogenes

Variety (Milk Reaction)	Basal Medium + Ca (1.5 mg./cc.)*	Basal Medium
Aureus	41 Orange	36 White 1 Yellow 4 Orange
Citreus	7 Yellow	6 White 1 Yellow
Albus	31 White	31 White

*Calcium was supplied as calcium gluconate. Cultures were incubated for 18 hours at 37° C. and 18 hours at 25° C.

examined. The pigment formation on the basal medium with calcium added corresponded in all cases with that on milk agar. Of the colored strains 42 showed no pigment in the absence of calcium while 6 did develop pigment. The albus varieties showed slight differences in pigmentation on the calcium-containing and on the calcium-free media. In the first case the colonies were porcelain white while in the latter case they appeared less opaque and were grayish in color.

Under the conditions of study calcium has a profound effect upon chromogenesis in *Micrococcus pyogenes*. The mode of action of this ion is not understood. Three sites of activity might be considered: (1) at the medium level where it might maintain suitable osmotic values or where it might form a gel which would absorb inhibitory substances from the medium; (2) at the membrane level where it might maintain or alter permeability; or (3) at the enzyme level where specific enzymes or systems might be activated or inhibited. In the first case both alternatives given seem improbable because chromogenesis is not stimulated by sodium or potassium chloride at osmotic concentrations equal to those of the active calcium solutions. The action of calcium as an adsorbent is ruled out by the experiment shown in table 2. The permeability of the cell membrane is undoubtedly altered

by calcium, but the experiments reported can offer no information on this point. The effect of calcium upon oxalacetate formation is of interest in connection with carotenoid biogenesis, at least from the speculative point of view. Calcium inhibits the formation and utilization of oxalacetate in animal preparations according to the experiments of Tyler (1955), and the inhibition of oxalacetate formation would cause the accumulation of acetate derived from the degradation of sugars and fats. It has been shown in plant systems by Bonner and Arreguin (1949) and in animal systems by Block and Rittenberg (1942) that acetate can be converted to isoprenoids. Consequently it is to be expected that accumulation of acetate would enhance the formation of isoprenoids such as the carotenoids.

Summary. Calcium is required for rapid chromogenesis in *Micrococcus pyogenes*.

Magnesium frequently antogonizes the effect of calcium upon chromogenesis.

The probable site of action of calcium is discussed.

REFERENCES

Altmen, S. M., and E. M. Crook: Activation of enzymes by chelating agents. *Nature*, 171: 76–77, 1953.

Block, K., and D. Rittenberg: On the utilization of acetic acid for cholesterol formation. *Jour. Biol. Chem.*, 145: 625–636, 1942.

Bonner, J., and B. Arreguin: The biochemistry of rubber formation in the Guayule. I. Rubber formation in seedlings. *Arch Biochem.*, 21: 109–124, 1949.

Fujita, A., and S. Yoshioka: A new medium favorable for pigment production by staphylococcus, also a contribution to the knowledge of pigment production. *Japan Med. World*, 3: 47–51, 1923.

Tyler, D. B.: Effect of ethylenediamine tetracetic acid, magnesium and calcium on succinate and keto acid metabolism. *Proc. Soc. Exptl. Biol. and Med.*, 88: 333–339, 1955.

Warner, R. C.: Separation of alpha and beta casein. *Jour. Am. Chem. Soc.*, 66: 1725–1731, 1944.

PARTICIPATION OF THE ALLERGEN-SPECIFIC ENZYMATIC FACTOR IN ALLERGIC SKIN REACTION*

EUGENE L. NOWICKI†

The mechanism of any immediate allergic reaction is still unknown. The theory of antigen-antibody reaction causing allergic manifestations was recently criticised by many allergists and immunologists. Not only allergic reactions but even some defense mechanisms do not fit into the antigen-antibody theory. Moulder (1948), Becker, and Gallagher (1947) discussing the defense mechanism against anthrax, and Taliaferro (1948) the antitrypanosomal factor (ablastin), suggested that it may be an oxidative enzyme in which panthothenic acid serves as the active prosthetic group. Sidney (1953) sees in the hemagglutinating action of some such viruses as mumps, influenza, and Newcastle, a similarity to an enzyme-substrate reaction. Godlowski (1953) theorizing about the mechanisms of anaphylaxis and allergy built up an enzymatic concept: the antigen or allergen penetrates into cells, and since the normal enzymes are not able to catabolize the antigen, a new "adaptive enzyme" is formed. This adaptive enzyme may be specific for the antigen and in some cases may be unable to carry out complete hydrolysis of the invading protein. The incomplete proteolysis may result in toxic products, for example, proteoses causing inflammatory reaction of the tissue. Dixon (1954) studying the metabolism of antigen labeled with I^{131} differentiates two phases in the disappearance of antigen: first a slow disappearance prior to antibody formation, and second, a faster disappearance when antibody reaches higher levels. The catabolism of antigen, even prior to antibody formation, is reflected in the appearance of non-protein bound I^{131}. Spies, Chambers et al. (1953) investigated the chemistry of allergens. Acting with various enzymes upon the allergenic fraction of cottonseed the authors stated: 1) That the skin reacting capacity of allergen was destroyed

* Department of Bacteriology and Parasitology, The University of Texas, Medical Branch, Galveston. Accepted for publication, October 27, 1955.
† Supported by J. McLaughlin Postdoctoral Fellowship.

by trypsin. 2) Decreased activity remained after digestion with chymotrypsin. 3) The skin-reacting capacity was not destroyed by pepsin or carboxypeptidase. 4) A dialysable fraction of the pepsin digest was slightly more potent in skin-reactivity than the nondialysable fraction. In the authors' conclusion, the intact protein was not essential for allergenic activity.

Recently doubts have appeared concerning the role of histamine in allergy. Noah and Brand (1955) incubated antigens of various origins with the blood of a sensitive subject. In some cases the antigen producing the largest skin reactions in a subject also causes the greatest release of histamine during incubation. In some instances, clinically significant antigens liberate large amounts of histamine when the skin tests are negative; in others, both skin test and release of blood histamine are negative when there is a good clinical history of sensitivity to an antigen.

The appearance of the specific enzymatic factor against an antigen was studied in several experiments (Nowicki, 1955). In the course of immunization against gamma globulin as well as against Leptospira, Salmonella, Shigella, and Staphylo- and Meningococcus, an enzymatic factor appeared in blood and was excreted in urine. For some antigens this enzymatic factor possesses a specificity well expressed, for others a wide group specificity. The activity of this enzymatic factor, fractionated and concentrated from urine, was described on the basis of various reactions *in vitro*.

The above mentioned experimental results as well as theoretical approaches led to the idea that the enzymatic factor which appears in the body in response to antigen introduction may take part in the allergic reaction.

Materials and Methods

In the experiments the purified gamma globulin was used as antigen. Antigen was introduced into guinea pigs, rabbits and mice by intracutaneous injection. The gamma globulin was used in 0.5 per cent solution for the first injection, and 0.05 per cent solution for succeeding injections. The enzymatic factor was previously produced in rabbits by intravenous injection of gamma globulins, and the urines from these rabbits were collected on days in which they were most active, in accordance with our previous work on enzymes in course of immunization (Nowicki, 1955a). In the present work urines were treated in the same way as described above, fractionated with acetone, redissolved in saline, to 1/10 of the original urine volume, and sterilized by filtration through a Masson filter. The experiments were concerned with two

factors. 1, gamma globulin, and 2, the urine fractions containing the enzymatic factor specific for the gamma globulin. Through a variety of injections into experimental animals the study of participation of the enzymatic factor in allergic reactions was carried out.

Results

The experiments were carried out in several steps which are presented in corresponding tables. Table 1 shows the result of

TABLE 1

Allergic skin reaction in five control rabbits. First intracutaneous injection of 0.2 ml. of 0.5 per cent gamma globulin on the left side of the abdominal wall. Second injection of 0.2 ml. of 0.05 per cent gamma globulin on the right side of the abdominal wall. Third injection same amount as the second one, on the left side

Side of injection	SKIN REACTION HOURS AFTER INJECTION 5	24	5	24	29
left	negative	negative	.....	.....	.....
right	.....	.....	left: erythema 15 mm. & edema	left: erythema 10 mm.	.....
left	.....	.....	.....	.....	both sides: erythema & edema

the control injections with gamma globulin in order to check the allergic activity of the substance. The solution of gamma globulin gave an allergic skin reaction after repeated intracutaneous injections. Similar results were obtained after sensitizing intracutaneous injections and the succeeding intravenous injections of gamma globulin (Table 2). The same solutions of

TABLE 2

Allergic skin reaction in five control rabbits. First injection intracutaneous of 0.2 ml. of 0.5 per cent gamma globulin. Second injection intravenous of 0.2 ml. of 0.05 per cent gamma globulin

	SKIN REACTION HOURS AFTER INJECTION 6	24	6	24
intracutaneous injection	negative	negative	.....	.....
intravenous injection	.....	.....	positive	positive

gamma globulin were used for further experiments. In table 3 are presented results of intracutaneous sensitization with gamma

TABLE 3

Skin reaction in five rabbits. First injection intracutaneous of 0.2 ml. of 0.5 per cent gamma globulin. Second and third intracutaneous injections of 0.05 per cent gamma globulin in urine fraction containing gamma globulin-specific enzymatic factor. Incubated for 16 hours at 37° C.

Side of injection	SKIN REACTION HOURS AFTER INJECTION				
	6	24	6	24	6
left	negative	negative			
right			negative	negative	
left					negative

globulin followed by intracutaneous injections of gamma globulin after an incubation period of 16 hours with the enzymatic factor specific for gamma globulin. The result shows, that after incubation the gamma globulin lost its antigenic property. No skin reaction appeared in rabbits after repeated intracutaneous injections. The same experiment was repeated on guinea pigs, as shown in tables 4 and 5. Repeated intracutaneous injections of gamma globulin produced a positive skin reaction (Table 4).

TABLE 4

Allergic skin reaction in five control guinea pigs. First intracutaneous injection of 0.2 ml. of 0.5 per cent gamma globulin on the left side of the abdominal wall. Second and succeeding injections of 0.2 ml. of 0.05 per cent gamma globulin

Side of injection	SKIN REACTION HOURS AFTER INJECTION					
	18	22	18	22	18	22
left						
right	neg.	neg.				
left			pos.	pos.		
right					pos.	pos.

After incubation with the enzymatic factor no skin reaction was visible in sensitized guinea pigs (Table 5).

In the following experiment 0.5 per cent gamma globulin solution (as used for sensitization) was mixed in equal parts with urine fraction containing the specific enzymatic factor, and immediately injected intracutaneously. Table 6 presents the

TABLE 5

Skin reaction in five guinea pigs. First injection intracutaneously of 0.2 ml. of 0.5 per cent gamma globulin. Second and succeeding injections of 0.2 ml. intracutaneously of 0.05 per cent gamma globulin diluted from 0.5 per cent solution with gamma globulin-specific enzymatic factor from urine, incubated for 16 hours at 37° C.

Side of injection	SKIN REACTION HOURS AFTER INJECTION					
	42	50	19	4	19	6
left						
right	neg.	neg.	neg.			
left				neg.	neg.	
right						neg.

results on rabbits, and table 7 on guinea pigs. In both species the positive skin reaction appeared in two to three hours after injection. The positive reaction appeared without the previous sensitization, after the first intracutaneous injection.

TABLE 6

Skin reaction in five rabbits. The first and the succeeding injections of mixture: 0.5 per cent gamma globulin and gamma globulin-specific enzymatic factor in equal parts (mixed in syringe)

Side of injection	SKIN REACTION HOURS AFTER INJECTION				
	4	23	4	24	4
left	positive (left)	positive (left)			
right			positive (left & right)	positive (left & right)	
left					stronger positive (left & right)

The same experiment was repeated again on a new set of guinea pigs, and the skin reaction was observed every hour for six hours and after twenty-two hours (the following day). The results are listed in table 8. In all guinea pigs (except No. 4) the erythema appeared around the point of injection after one hour. In two hours time the erythema was 11 mm. in diameter, and the edema was prominent. After three hours the erythema reached the size of 14 mm., and the edema was considerable.

TABLE 7

Skin reaction of five guinea pigs. The first and the succeeding injections of mixture: 0.5 per cent gamma globulin and gamma globulin-specific enzymatic factor in equal parts (mixed in syringe)

Side of injection	SKIN REACTION HOURS AFTER INJECTION				
	3	24	4	24	4
left	positive (left)	positive (left)	.....	.....	.....
right	.....	.....	positive (left & right)	positive (left & right)	.....
left	.....	.....	.....	.....	stronger positive (left & right)

This reaction persisted without change for the following three hours. After twenty two hours (the following day) the erythema was still visible, and mild edema was present. This experiment shows that the skin reaction appears earlier when gamma globulin is injected together with the enzymatic factor in non-sensitized skin than it does during the usual procedure (sensitizing gamma globulin injection followed by injections of gamma globulin). The duration of the reaction is the same in both cases.

Comparing the results of tables 3 and 5 with tables 6, 7, and 8 it is evident that gamma globulin loses antigenic (allergenic) properties after incubation with the specific enzymatic factor at 37° C. for 16 hours. On the other hand, gamma globulin acquires a new property of giving a positive skin reaction in non-sensitized animals when injected immediately after mixing with the specific proteolytic factor. Consequently, the following experiment was concerned with the time required for changing the property of the mentioned mixture (gamma globulin plus enzymatic factor). Gamma globulin solution, 0.5 per cent, was mixed in equal parts with the urine fraction containing the enzymatic factor, and placed at 37° C. Every hour some (0.15 ml.) of this liquid was injected intracutaneously in another guinea pig. The skin reaction was observed every half hour for five hours, then after 20 hours (next day). The results are presented in table 9. After one hour of incubation the injected material gave a skin reaction identical, within 5 hours, with the reaction listed in table 8. After 20 hours the skin reaction disappeared, therefore

it was a little weaker than in table 8. The mixture injected after two hours of incubation gave a weak reaction which started after thirty minutes and lasted for three hours. After three, four, and five hours of incubation there was no skin reaction or mild erythema for a short period. After 20 hours they were all negative. The above experiment shows that the change of gamma globulin starts after one hour of incubation: the skin reaction appears earlier (after thirty minutes), and disappears earlier (before 20 hours). After two hours of incubation the change of gamma globulin is more advanced: in thirty minutes time the positive skin reaction is well expressed but disappears after three hours. An incubation period of three hours is sufficient to split the gamma globulin so that the skin reaction becomes negative. The above mentioned time of splitting can be applied only to this particular experiment. This time depends upon concentration of antigen solution and upon the activity of the enzymatic factor. Nevertheless, the experiment demonstrates the relation between the allergenic property of antigen and the specific enzymatic factor.

TABLE 8

0.5 per cent gamma globulin solution was mixed in syringe in equal parts with the enzymatic factor in urine fraction. Five guinea pigs were injected intracutaneously and the skin reaction observed every hour for six hours

Guinea pig No.	SKIN REACTION HOURS AFTER INJECTION						
	1	2	3	4	5	6	next day
1	mild eryth.	eryth. – 11 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	mild eryth., edema
2	mild eryth.	eryth. 11 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. and edema
3	mild eryth.	eryth. 11 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	mild eryth., & edema
4	no reaction	mild eryth.	no reaction	no reaction	no reaction	no reaction	no reaction
5	mild eryth.	eryth. 11 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	mild eryth., & edema

Several experiments were carried out on mice. The reaction of control animals was not uniform, therefore the results were not evaluated.

The problem of the specificity of the reaction between the enzymatic factor and the antigen was described and discussed largely in previous publications (Nowicki, 1955). Identical results were obtained in experimenting with the specificity of the enzymatic factor-allergen reaction as it was described for various antigens used for immunization in "Studies on enzymatic factor in course of immunization." For this reason the specificity is not again discussed. Concerning the allergenic activity of the urine fraction, the tables 3 and 5 give the answer: the enzymatic factor in urine fraction has no allergenic activity.

TABLE 9

0.5 per cent gamma globulin mixed in equal parts with the enzymatic factor in urine fraction. The mixture kept at 37° C., and 0.15 ml. of the mixture injected intracutaneously to normal guinea pigs every hour

Hours of incubation	SKIN REACTION HOURS AFTER INJECTION ½	1	1½	2	2½	3	3½	4	4½	5	20
1	no react.	mild eryth.	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	eryth. 14 mm., edema	mild eryth. edema	mild eryth. edema	no react.
2	eryth. 14 mm., edema	eryth. 14 mm., edema	mild eryth. edema	eryth. edema	eryth. edema	eryth. and edema	eryth. and edema	....	....	....	no react.
3	no react.	no react.	no react.	no react.	no react.	no react.					no react.
4	no react.	no react.	no react.	no react.	no react.	....					no react.
5	no react.	no react.	no react.	no react.	....	....					no react.

Discussion. In previous publications it was shown that the injection of antigen produces a specific enzymatic factor which remains in the organism for several days. It was a theoretical possibility that this antigen-specific enzymatic factor takes a certain part in the allergic reaction produced by the second or succeeding injections of the antigen. Previous experiments have shown that the enzymatic factor can split the antigen *in vitro*. This was confirmed in the first series of experiments presented in this

paper: the second and the succeeding injections of antigen previously treated with specific enzymatic factor do not produce any allergic skin reaction.

The second series of experiments showed that the action of the specific enzymatic factor upon the antigen is an important component in allergic reaction. The mixture of the antigen and the specific enzymatic factor, prepared in a syringe, produces an inflammatory skin reaction in about two hours after the *first* injection. It shows the importance of the enzymatic factor for the allergic skin reaction in general. If the antigen is injected together with the specific enzymatic factor, the skin reaction appears without previous sensitization. The logical conclusion of this result is that, in normal allergic skin reaction, the first injection of antigen produces in the body the antigen specific enzymatic factor, which accelerates the decomposition of antigen introduced in the second or in the succeeding injections. Probably products of this enzymatic decomposition of the antigen cause the inflammatory reaction. This may give an idea of at least one factor of the shock reaction: the antigen specific enzymatic factor must be produced by some tissue (organs), where it is in higher concentration than in the blood stream. When this enzymatic factor is present, and the further intravenous or intramuscular introduction of antigen follows, it results in a rapid decomposition of antigen in organs leading to inflammatory reaction. It is an interesting working hypothesis to study the concentration of enzymatic factor in different organs after introduction of sensitizing antigen and study further the relation of this concentration to the shock reaction. As the shock reaction is an unstable manifestation probably the ability of tissue to retain the enzymatic factor is an individual one.

Another conclusion can be drawn from our experiments: the allergic skin reaction is produced by the products of the early splitting of antigen. The facts leading to this conclusion are: when the enzymatic factor is allowed to act upon the antigen for several hours (more than 3 in table 9) at 37° C., the injection of this mixture does not produce an inflammatory reaction. Only after the simultaneous introduction of antigen and enzymatic factor or after previous incubation of less than 2 hours, the inflammatory reaction appears shortly after injection. The short

incubation causes an earlier appearance of the skin reaction. Therefore, it is probable that some polypeptides and not the aminoacids, are responsible for the inflammatory reaction.

A further study of enzymatic factor in allergic reactions may give some new ideas in the still obscure problem of allergy, as well as of shock.

The intracutaneous injection of guinea pigs with the mixture of antigen and enzymatic factor, seems to give a simple and fast method of showing the presence of the specific enzymatic factor in the organism. This reaction could be used for checking a sensitized condition: it is more sensitive and more specific than the usual skin test. Another working hypothesis of interest is the possible application of the reaction for clinical diagnosis instead of incubation with coagulated tissue followed by ninhydrin reaction.

Summary. An enzymatic factor was fractioned and concentrated from urine of rabbits injected intravenously with gamma globulin. The urine fraction containing the gamma globulin specific enzymatic factor was used in various combinations with gamma globulin for intradermal injections in guinea pigs, rabbits and mice. The results can be summarized as follows:

1) Gamma globulin was mixed with the enzymatic factor. After incubation of the mixture at 37° C. for 16 hours, the antigen was catabolized to such a degree that it lost its antigenic property, and did not produce a skin reaction in sensitized animals. 2) When antigen was injected together with the enzymatic factor in unsensitized animals the mixture caused an inflammatory skin reaction. 3) The antigen was mixed with the enzymatic factor and kept at 37° C. Every hour some of the mixture was injected in normal guinea pigs. After one hour of incubation the skin reaction appeared the same as when injected immediately. After two hours the skin reaction appeared earlier and was weaker, and after three or more hours there was no skin reaction. The conclusion is that the enzymatic factor is in part responsible for the allergic skin reaction, and that the products of early catabolism of the antigen are responsible for the inflammatory reaction of the skin.

REFERENCES

Becker, E. R., and P. L. Gallagher: Prolongment of the Productive Phase of Trypanosoma Lewisi by the Administration of Sodium Salicylate. *Iowa State Coll. J. Sci.*, 21: 351–361, 1947.

Dixon, F. I.: The Metabolism of Antigen and Antibody. *J. Allergy*, 25: 487–503, 1954.

Godlowski, Z. L.: *Enzymatic Concept of Anaphylaxis and Allergy*. E. S. Livingstone Ltd., Edinburgh and London, 1953.

Moulder, J.: Changes in the Glucose Metabolism of Trypanosoma Lewisi during the Course of Infection in the Rat. *J. Inf. Dis.*, 83: 42–49, 1948.

Noah, J. W., and A. Brand: Correlation of Blood Histamine Release and Skin Test Response to Multiple Antigens. *J. Allergy*, 26: 385–393, 1955.

Nowicki, E. L.: Early Diagnosis of Leptospirosis by Lysis-Test with Urine Fractions. *Texas Rep.Biol.Med.*, 13: 293–305, 1955.

Nowicki, E. L.: Studies on Enzymatic Factor in Course of Immunization. *Texas Rep.Biol.Med.*, 13: 848–864, 1955a.

Nowicki, E. L.: Early Diagnosis of Salmonella, Shigella, and Coccus Infections by Lysis-Test with Urine Fractions. *Texas Rep.Biol.Med.*, 13: 840–847, 1955b.

Taliaferro, W. H.: The Inhibition of Reproduction of Parasites by Immune Factors. *Bact.Rev.*, 12: 1–17, 1948.

Sheldon, J. M., R. G. Lovell, and K. P. Mathews: *Clinical Allergy*. Philadelphia, Saunders, 1953.

Sidney, R.: *Immunity*. New York, Appleton-Century-Crofts, 1953.

Spies, J. R., D. C. Chambers, and et al.: The Chemistry of Allergens. XII. Proteolysis of the Cottonseed Allergen. *J. Allergy*, 24: 483–491, 1953.

CALLING ATTENTION TO

OHIO STATE UNIVERSITY, COLUMBUS 10, OHIO

NOVEMBER, 1955

Of General Interest: JW Bell rings the Bells (John 1763–1825 & Charles 1774–1842) of Edinburgh (*Surgery* 38/794/55). . . . JW Black & WE Moore give good analysis of *Speech: Code, Meaning, & Communication* (McGraw-Hill, NY, 55, 427pp, $4.5). . . . FJ Brown & T Sellin edit important symposium on *Higher Education under Stress* (*Ann. Amer Acad Pol Soc Sci* 301/1–210/Sept 55). . . . N Cousins, power to his pen, gives interim report on plastic & psychological repair of famed "Hiroshima Maidens" (*Sat Rev* Oct 15/55, p22). . . . CD Curtis finds our government harms us all by security rules such as operated in *The Oppenheimer Case* (Simon & Schuster, NY, 55, 281pp, $4). . . . MR Harrower well edits *Medical & Psychological Team-Work in the Care of the Chronically Ill*, by 30 collaborators (CC Thomas, Springfield, Ill, 55, 232pp, $4). . . . P Jordan reflects on *Science & the Course of History: The Influence of Scientific Research on Human Events* (Yale Univ Press, New Haven, Conn, 55, 139pp, $2.5). . . . D Keene compiles *An Anthology of Japanese Literature* (Grove Press, NY, 55, 480pp, $6.5). . . . WT Pommerenke writes well on abortion in Japan (*Obs Gyn Survey* 10/145–175/55). . . . WD Postell fills need by *Applied Medical Bibliography for Students* (CC Thomas, Springfield, Ill, 55, 142pp, $4). G Sarton, as usual, is superb as Rosenbach Fellow in discussing *The Appreciation of Ancient & Medieval Science during the Renaissance* 1450–1600 (Univ Pennsylvania Press, Phila, 55, 223pp, $6). . . . H Selye reviews stress & disease (*Science* 122/ 625/Oct 7/55). . . .EW Sinnott considers *The Biology of the Spirit* (Viking Press, NY, 55, 180pp, $3.5). . . . WG Smillie looks forward from backward on *Public Health: Its Promise for the Future: A Chronicle of the Development of Public Health in the United States 1607–1914* (Macmillan, NY, 55, 501pp, $7.5). . . . L Untermeyer tells about *Makers of the Modern World* (Simon & Shuster, NY, 55, 809pp, $6.5). . . . K Walker is inaccurately enthusiastic about *The Story of Medicine* (Oxford Univ Press, NY, 55, 343pp, $6). . . . I Wallace enjoys *The Fabulous Originals: Lives of Extraordinary People Who Inspired Memorable Characters in Fiction* (AA Knopf, NY, 55, 317pp, $4).

Of Medical Interest: TK Adler & Co show codeine is half excreted in 6 hours, with biotransformation by demethylation (*J Pharmacol* 114/251/55). . . . TM Brody suggests uncoupling of oxidative phosphorylation as factor in drug action (*Pharmacol Rev* 7/335/55). . . . JR Elkinton & TS Danowski well survey *The Body Fluids* (Williams & Wilkins, Baltimore, Md, 55, 600 pp, $10). . . . E Flory & H McLennan find dialysable heat-stable brain extractive which inhibits synaptic transmission (*J Physiol* 129/384/55). . . . JD Hardy & Co continue studies on thermal burns in humans (*Surgery* 38/685, 692/55). . . . GM Hocking prepares useful *Dictionary of Terms in Pharmacognosy & Economic Botany* (CC Thomas, Springfield, Ill, 55, 284pp, $6). . . .A Hollaender

edits monumental *Radiation Biology* (McGraw-Hill, NY, 55, 3 vols, 2365pp, $34). . . . EA Hosein & Co show nitrous oxide inhibits oxygen consumption of brain (*Anesth* 16/708/55). . . . FP Luduena finds dithylaminoethyl-2-propoxy-4-aminobenzoate ("ravocaine") a potent rapidly diffusing non-irritating local anesthetic (*Anesth* 16/751/55). . . . CG Peterson analyses neuropharmacology of procaine (*ibid* p 678). . . . L Pillemer & Co discuss interaction of properdin system & polysaccharides in relation to immunity (*Science* 122/545/Sept 23/55). . . . WC Randall & KK Kimura analyse pharmacology of sweating (*Pharmacol Rev* 7/365/55). . . . C Rimington reviews porphyrins (*Endeavor* 14/126/55). . . . M Rocha y Silva discusses *Histamine: Its Role in Anaphylaxis & Allergy* (CC Thomas, Springfield, Ill, 55, 248pp, $5). . . . A Roe surveys metabolism & toxicity of methanol (*Pharmacol Rev* 7/399/55). . . . RJ Shephard finds breathing CO_2 mixtures increases oxygen consumption & heart output (*J Physiol* 129/393/55). . . . V Trendelenburg finds histamine & pilocarpine potentiate ganglionic transmission (*J Physiol* 129/337/55). . . . WP Vanderlaan & VM Storrie discuss antithyroid agents (*Pharmacol Rev* 7/301/55). . . . A Vogl reviews *Diuretic Therapy: The Pharmacology of Diuretic Agents & the Clinical Management of Edematous Patients* (Williams & Wilkins, Baltimore, Md, 55, 255pp, $5). . . . RP Walton & Co note toxic actions of decaborane ($B_{10}H_{14}$), a high energy gasoline potentiator, which causes blood cell clumping & bradycardia (*J Pharmacol* 114/367/55).

DECEMBER, 1955

Year-odds & ends: American Foundation offers *Medical Research: A Mid-century Survey* (Little Brown, Boston, 55, 2 vols, $15). . . . W Andrews well relates *Architecture, Ambition & Americans* (Harper, NY, 55, 315pp, $7.5). . . . H F Blum observes perspectives in evolution (*Amer Sci* 43/595/55). . . . I Bry & J Doe well document Albert Einstein's long effort to get scientists to renounce war (*Science* 122/911/Nov11/55). . . . R Carson finds much at *The Edge of the Sea* (Houghton Mifflin, Boston, 55, 276pp, $4). . . . TE Gaddis, solitarily confined lifer, becomes expert ornithologist in *Birdman of Alcatraz* (Random House, NY, 55, 265pp, $4). . . . WC Muenscher & MA Rice like *Garden Spice & Wild Pot-Herbs* (Cornell Univ Press, Ithaca, NY, 55, 218pp, $5.75). . . . R Hofstadter & WP Metzger trace *The Development of Academic Freedom in the United States* (Columbia Univ Press, NY, 55, 527pp, $5.5). . . . A Pi Suner analyses *Classics of Biology* (Philosophical Library, NY, 55, 322pp, $7.5). . . . AH Snell wrote exciting Geneva Diary for Aug 8–20/55 (*Amer Sci* 43/505/55). . . . J Thurber releases *Thurber's Dogs* (Simon & Schuster, NY, 55, 210pp, $4). . . . Chief Justice Earl Warren looks with wisdom on the law & the future (*Fortune* 52/No 5/Nov 55/ p 106). . . . WH Whyte Jr considers what the big Foundations are up to, & indicates the kind of oil to use to get grants (*Ibid* Oct/55 p110). . . . E Wilson describes the fascinating *Scrolls from the Dead Sea* (Oxford Univ Press, Ny, 55, 168pp, $3.25).

The Skin Gets a Break: AC Allen produces a great tome in *The Skin: A Clinicopathologic Treatise* (CV Mosby Co, St.Louis, 55, 1048pp, $20). . . . K Hellman finds cholinesterase in all skin structures with cholinergic in-

nervation (like human eccrine sweat glands), & non-specific pseudocholinesterase in arrector pili muscles in human skin (*J Physiol* 129/454/55). . . . S Rothman's *Physiology & Biochemistry of the Skin* is a real classic (Univ Chicago Press, 55, 741pp, $19.5).

Cancer Smoke: RL Cooper & AJ Lindsey extract 4 micrograms of carcinogen 3,4-benzpyrene from 500 cigarettes (*Brit J Cancer* 9/304/55). . . . P Stocks & JM Campbell suggest benzpyrene plays dual role in lung cancer through cigarettes & smoke pollution of air in cities (*Brit Med J* 2/923/Oct 15/55). *Clinical:* JH Comroe & Co consider *The Lung: Clinical Physiology & Pulmonary Function Tests* (Yearbook Publ, Chicago, 55, 219pp, $5.5). . . . P Knocker shows anoxic histopathology in hypothermia (*Lancet* 2/837/Oct 22/55). . . . S Standish & Co study *Why Patients See Doctors* (Univ Washington Press, Seattle, 55, 176pp, $2.5). . . . DB Tyler notes psychotic behavior in experimental sleep deprivation (Dis Nerv Syst 16/293/55). . . . PD White issues timely *Clues in the Diagnosis & Treatment of Heart Disease* (CC Thomas, Springfield, Ill, 55, 192pp, $5.5).

Preclinical: HA Abramson & Co offer many keen reports on lysergic acid diethylamide psychobiological effects (*J Psych* 39/3, 127, 373, 435, 443, 455, 465/55). . . . (*Ibid* 40/39, 53, 337, 341, 351, 367, 385/55!!). . . . GE Anderson describes glucagon as regulator of insulin function (*Science* 122/457/Sept 9/55). . . . BB Brodie & Co indicate neurohumoral function of 5-hydroxytryptamine in brain (*Science* 122/968/Nov 18/55). . . . M Grunberg-Manago & Co review enzymatic synthesis of polynucleotides (*Science* 122/907/Nov 11/55). . . . PG LeFevre reviews *Active Transport through Animal Membranes* (Protoplasmatologia, Vol 8, Physiologie des Protoplasmas, Lange & Springer, Berlin, 55, 140pp, $9). KF Meyer has a new one: *The Zoonoses in Their Relation to Rural Health* (Univ California Press, Berkeley, 55, 54pp, $1). . . . P Nordquist & P Wistrand show acetazolamide (Diamox) increases stimulation threshold of peripheral nerves due to hyperpolarization (*Acta Soc Med Upsala* 60/56/55). . . . AH Owens Jr & EK Marshall Jr find variation in metabolic fate of chloral hydrate & tricholrethanol from urinary excretion of trichloracetic acid to biliary excretion of trichlorethanol glucuronide (*Bull Johns Hopk Hosp* 97/320/55). CP Whittingham discusses photosynthesis (*Endeavor* 14/173/55). . . .

JANUARY, 1956

New Year General: MM Belli completes *Modern Trials* (Bobbs-Merrill, Indianapolis, 55, 3 vols, $60.). S. Dickson shows *San Francisco Profiles* (Stanford Univ Press, 55, 3 vols, $10). RS Kirby & Co survey *Engineering in History* (McGraw-Hill, NY, 56, 686pp, $7). H Marcuse philosophizes on Freudian principles in *Eros & Civilization*, visualizing a non-repressed culture culture (Beacon Press, Boston, 55, 277pp, $4). AT Mason discusses *Security through Freedom* (Cornell Univ Press, Ithaca, NY, 55, 243pp, $2.9). JR Newman edits essays on *What is Science?* (Simon & Schuster, NY, 55, 493pp, $5). JR Oppenheimer well discourses on The Open Mind (Simon & Schuster, NY, 55, 146pp, $2.75). BB Patterson reminds us of obligations of freedom in *The Forgotten Ninth Amendment* (Bobbs-Merrill, Indianapolis, 55, 228pp, $4).

PB Rice writes well *On the Knowledge of Good & Evil* (Random House, NY, 55, 299pp, $5).

Addiction Problems: JL Free considers *Just One More: Concerning the Problem Drinker* (Coward-McCann, NY, 55, 210pp, $3.5). H. Isbell is hopeful about medical aspects of opiate addiction, with number declining (*Bull NY Acad Med* 31/886/55). M. Wellman suggests environmental factors in social conformity among strong young men as factor in alcoholism (*Canad Med Asso J* 73/717/55).

Preclinical: AS Dontas show chemoreceptors of carotid body are stimulated by high energy phosphates & dinitrophenol, & depressed by barbitals & glucose (*J Pharmacol* 115/46/55). THJ Huisman & Co note Hb-C has higher lysine & histidine content & is more basic than Hbs A, S, or F (*Blood* 10/1079/55). FH Johnson edits *Luminescence of Biological Systems* (AAAS, Washington DC, 55, 452pp, $4.5). AM Lands & Co find bisquaternary oxamides synaptically active as specific antagonists of acetylcholinesterase, d-tubocurarine & tetraethyl pyrophosphate (*J Pharmacol* 115/185/55). GJ Martin outlines *Biological Antagonisms: The Theory of Biological Relativity* (McGraw-Hill, NY, 55, 516pp $9). HJ McDonald tells about *Ionography: Electrophoresis in Stabilized Media* (YearBook, Chicago, 55, 268pp, $6.5). K Okamoto gets spontaneous diabetes by successive breeding of rabbits with alloxan-induced diabetes (*Tohuku J Exp Med* 61/ Suppl II/ 116pp/ 55). E Ponder *Red Cell Structure* & Its Breakdown (Vol 10, Protoplasmatologia, Lange & Springer, Berlin, 55, 140pp, $9.5). Our LA Sapirstein & Co note that failing blood circulation as in hemorrhage tends to support splanchnic bed, after reserve discharge (*Science* 122/1138/Dec 9/55). EF Scowen & G Hadfield note hypophyseal agent in premenopausal urine able to cause mammary duct proliferation (*Cancer* 8/890/55). AC Upton considers pathogenesis of hemorrhagic state in radiation illness (*Blood* 10/1156/55).

Clinical: D Adlersberg considers inborn errors of lipid metabolism (*Arch Path* 60/481/55). CA Boyd describes *Autoradiography in Biology & Medicine* (Academic Press, NY, 55, 411pp, $8.8). WH Cole offers *Some Physiological Aspects & Consequences of Parasitism* (Rutgers Univ Press, New Brunswick, NJ, 55, 90pp, $2). P DeNicola gives *The Laboratory Diagnosis of Coagulation Defects* (CC Thomas, Springfield, Ill, 56, 228pp, $6). JE Dunphy & Co show high protein & ascorbic acid necessary for normal wound healing (*New Eng J Med* 253/847/Nov 17/55). DAG Galton & Co find water sol p(di-2-chlorethylamino) phenylbutyric acid gives remissions in malignant lymphomas at oral dose of 0.2mg/Kg with little gastroenteric effects or hematopoetic damage (*Brit Med J* 2/1172/Nov 12/55). MF Heller offers *Functional Etology: The Practice of Audiology* (Springer, NY, 55, 225pp, $5.5). JO Hoppe & Co show ferrous gluconate safest & most effective for iron therapy (*Am J Med Sci* 230/491/55). A Kerr discusses *Subacute Bacterial Endocarditis* (CC Thomas, Springfield, Ill, 55, 343pp, $6.). RF Sognnaes edits *Advances in Experimental Caries Research* (AAAS, Washington DC, 55, 235pp, $5). H Viets & GD Gammon edit comprehensive symposium on myasthenia gravis (*Am J Med* 19/655–742/55).

C. D. Leake